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COARCTATION OF THE AORTA: THE COLLATERAL CIRCULATION

BY

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From the Cardiographic Department, Manchester Royal Infirmary

Received June 9, 1941

Coarctation of the aorta imposes a mechanical embarrassment on the circulation and so affords an opportunity of studying problems of both clinical and physiological interest. During the past ten years we have had the good fortune to meet with thirteen patients exhibiting this abnormality, and in the present and future papers it is our object to consider some of the problems presented by this condition.

This paper is concerned with one of our patients in whom the lesion was diagnosed in 1938. Two years later she was admitted to hospital suffering from subarachnoid hæmorrhage, from which she died. This accident gave us the opportunity of studying the collateral circulation, in a patient who exhibited no signs of heart failure, by a method to which we have been able to find only one reference (Ernstone and Robins, 1931). We injected the arterial system with barium paste and took a series of radiograms of the cadaver.

CASE RECORD

A nulliparous woman, aged 25, consulted one of us (C. B.) in July 1938 regarding her fitness for pregnancy.

Following rheumatism at the age of 5, a heart murmur had been discovered. At 7 years she was troubled by growing pains. She suffered from chilblains and from cold hands and feet. At school she was never able to play games, and at the age of 15 was off school for a year with dyspnœa and palpitation. In childhood she suffered from migraine, which occurred almost every week and was associated with vomiting and severe bilateral frontal headache; this ceased in adult life. Between the ages of 17 and 24 she worked in an office, and during this period was never absent owing to illness. She gave up this work on marriage, nine months before we saw her.

At that time her chief complaints were of pain below the left scapula, which became worse on exertion, and of occasional dyspnœa, palpitation, and flatulent dyspepsia. Nevertheless she was able to do all her own housework with the help of a woman on one half-day each week.

On physical examination the first thing that struck one was the violent arterial pulsation in the neck. The pulse rate was 112 and its rhythm regular; the blood pressure was 235/100 in both arms. On auscultation there was a systolic murmur with an unusual distribution; it was heard both at the apex and at the base of the heart, but was loudest on the back of the chest on the right side, just above the spine of the scapula. The femoral pulse was of small volume, and the systolic blood pressure at the ankle was 140.

A diagnosis of coarctation of the aorta was made, and was confirmed by radiography (Fig. 1–3). This showed the characteristic notching of the ribs and the absence of the aortic knuckle (Fig. 1); and, in the right oblique position, the absence of the typical aortic impression on the barium-filled œsophagus (Fig. 2). In the electrocardiogram the T wave was diphasic in lead II and negative in lead III. A blood count showed hypochromic anæmia with 70 per cent hæmoglobin. The urine contained a trace of albumin; the specific gravity was 1018 and the renal function tests were satisfactory.

Apart from the pain in the back she remained fairly well until the morning of July 30, 1940, when suddenly she was seized by a severe pain in the occipital region; this spread down the back to the legs. She vomited four or five times, became semi-conscious, and subsequently spoke only once to complain of very severe headache. On admission to the Manchester Royal Infirmary that afternoon she was comatose, with signs of severe meningeal irritation. Her pulse was 80, full and bounding, and her blood pressure was 220/115. The left arm and leg were more flaccid than the right, and the left plantar response was extensor. The condition was diagnosed as subarachnoid hæmorrhage, and this diagnosis was confirmed by the withdrawal of a heavily blood-stained cerebro-spinal fluid under increased pressure. She died a few hours later.

Post-mortem Examination

(a) *Morbid Anatomy*. An autopsy was performed by Dr. W. Susman seventeen hours after death. Apart from œdema of the lower lobes of the lungs and slight generalized passive congestion, the abnormalities found were confined to the brain and cardiovascular system.

The brain weighed 1300 grammes. Blood was present in the subarachnoid space over the right cerebral hemisphere and the pons. A pressure cone was evident and the convolutions were flattened. Most of the outer half of the right parietal lobe had been destroyed by hæmorrhage, and the area was occupied by blood clot; the hæmorrhage had burst into the right lateral ventricle and blood was present in the right lateral, third, and fourth ventricles. In the plane of the pituitary stalk, just below the level of the optic thalamus, there was an aneurysm of the right middle cerebral artery, 0·8 cm. in diameter (Fig. 4). This aneurysm had recently ruptured.

The descending thoracic and the abdominal aorta were rather small. The intercostal arteries, the internal mammary arteries, and the arteries of the head, neck, and upper limbs were enlarged and tortuous.

R L

FIG. 1.—*Teleradiogram of the chest (Postero-Anterior).* The aortic knuckle is not visible, and there is notching of several ribs (see Fig. 17). The heart is not enlarged.

R L

FIG. 2.—*Teleradiogram of the chest in the right anterior (I) oblique position.* There is no typical aortic impression on the œsophagus; the posterior mediastinum is clear, and the œsophagus is not displaced by the left auricle.

R L

FIG. 3.—*Teleradiogram of the chest in the left anterior (II) oblique position.*

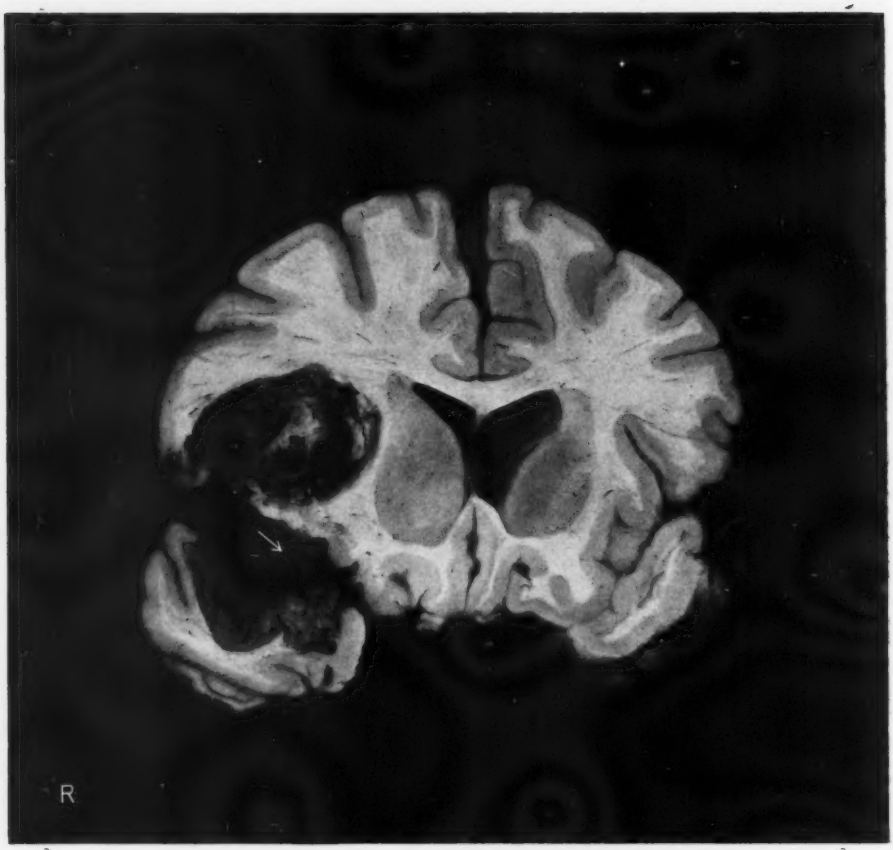


FIG. 4.—*Coronal section of the brain.* There is a large hæmorrhage in the right parietal lobe. The ruptured congenital aneurysm has been cut across and is indicated by an arrow. This association of congenital cerebral aneurysms with coarctation of the aorta has been noted by various authors (Brown, 1939).

The aorta appeared to be completely occluded at a point on the arch just distal to the left subclavian and near the entrance of the obliterated ductus arteriosus, which was represented by a stout fibrous strand (Fig. 5). The left subclavian artery arose as a direct continuation of the aortic arch and was unusually large. The upper intercostal arteries, entering the descending aorta just distal to the occlusion, were greatly dilated.

The heart weighed 350 grammes. The specimen, unfortunately, was destroyed, as the result of enemy action, before any further examination had been made.

(b) *Injection of the Arteries.* Prior to autopsy it was decided to attempt to inject the arteries of the intact cadaver with an opaque paste in order to

FIG. 5.—*The heart.* The coarctation is visible just distal to the origin of the left subclavian, which forms the continuation of the aortic arch. The innominate, the left common carotid, the left subclavian, and their branches are greatly enlarged. The upper aortic intercostals are seen cut across and are greatly enlarged. The obliterated ductus arteriosus persists as a fibrous cord.

study the collateral circulation. Barium enema paste was used, as it was readily available at short notice.

An incision was made on the left side of the neck and the left common carotid artery exposed; a canula was introduced and the barium injected slowly into the arch of the aorta, proximal to the coarctation, under radioscopic control. Films were taken at intervals in the anterior and oblique positions (Fig. 6, 7, 9, 10, and 11) and of the abdominal vessels (Fig. 12). It was found best to keep a moderate pressure on the piston of the syringe during the exposure in order to distend the aorta slightly.

It is important to inject the barium under radioscopic control, in order to ensure that sufficient enters the arteries to outline them satisfactorily without obscuring the larger vessels by filling the arterioles and capillaries. Flooding of the vessels occurs earlier in some areas than in others and it is advisable to take a series of films at different stages of the injection in order to display all the main vessels.

Between 700 and 800 c.c. of barium paste were used in this case. The paste used for barium enemata is suitable for this purpose; a thinner paste is liable to cause flooding before the main vessels are satisfactorily outlined.

The barium was injected above the coarctation, and therefore reached the vessels of the lower part of the body by the collateral channels. The descending aorta, the aortic intercostals, and the abdominal vessels were filled in this way. Since the barium followed the path taken by the blood during life, filling artefacts were avoided.

The results of the injection are shown in Fig. 6–11 which follow, and they are explained in the legends below each.

The first two show an early and late stage of the injection in the ordinary antero-posterior position. The injection canula is seen in the left common carotid. In Fig. 6 the intercostal arteries are only beginning to fill, but in Fig. 7 they are well filled and the tortuosities and notching of the ribs are clearly seen. The coarctation can be seen to the left of the mediastinal shadow and in Fig. 7 the tortuous enlarged subscapular artery can be seen on the right border of the chest. Fig. 8 is a tracing from Fig. 7, naming the arteries that can be seen.

Fig. 9 and 10 show the left (II) oblique positions: in both the coarctation is clearly seen and seems to be complete; in the former it is crossed by two tortuous and enlarged aortic intercostals passing upwards to anastomose with the superior intercostal. In Fig. 11 a clear picture has been taken of the main branches above the coarctation and of some from below (shown in darker shading).

Finally, Fig. 12 shows the abdominal vessels, which must have filled entirely through the collateral circulation, although the well-known anastomosis between the superior and deep epigastric arteries was not developed in this case. The legends below each figure give fuller details.

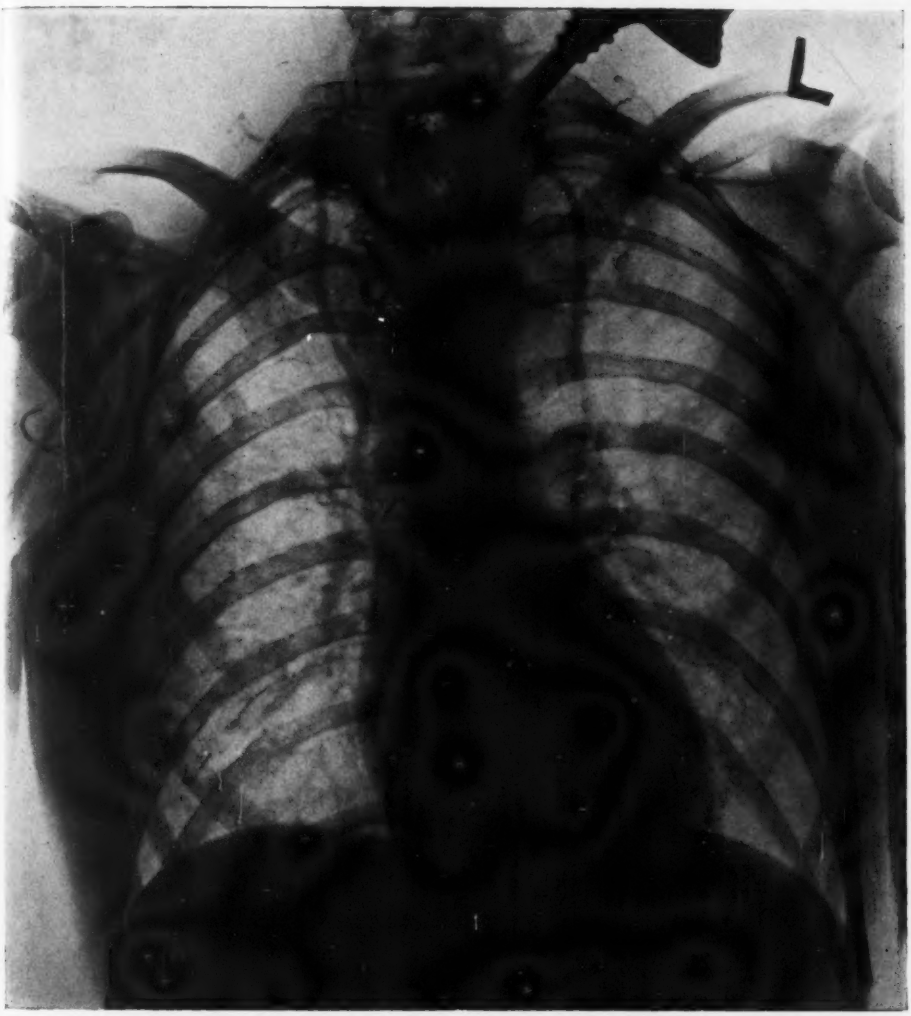


FIG. 6.—*Barium injection: Radiogram (P.-A.) at an early stage of injection.* The injection canula is seen in the left common carotid artery. The intercostals are commencing to fill by their anastomotic channels. The coarctation is shown to the left of the superior mediastinal shadow; the upper prominence is formed by the tortuous origin of the left subclavian (Fig. 5) and the lower prominence by the blind end of the descending aorta. Between this and the ascending aorta there is a narrow gap.

FIG. 7.—*Barium injection: Radiogram (P.-A.) at the end of injection.* The arteries shown on this radiogram are named in Fig. 8. The intercostal arteries have filled and are very tortuous. The notching of the ribs by the arterial loops is obvious. The internal mammary arteries are enlarged and the extensive diaphragmatic anastomosis formed by their musculophrenic branches and the phrenic branches of the aorta is well seen on the left side. The tortuous enlarged subscapular artery is visible on the right border of the chest; its dorsalis scapulæ branch encircles the neck of the scapula. Just medial to its origin is the long thoracic artery. The aortic intercostals supplying the fourth and fifth spaces are much enlarged near their origin from the aorta. This appearance is not a filling artefact, for all the barium was injected above the coarctation. The intercostals, therefore, filled by their collateral channels from the scapular network and the superior intercostal, and the distension near the descending aorta could not have been due to forcing barium into them from the aorta distal to the coarctation.

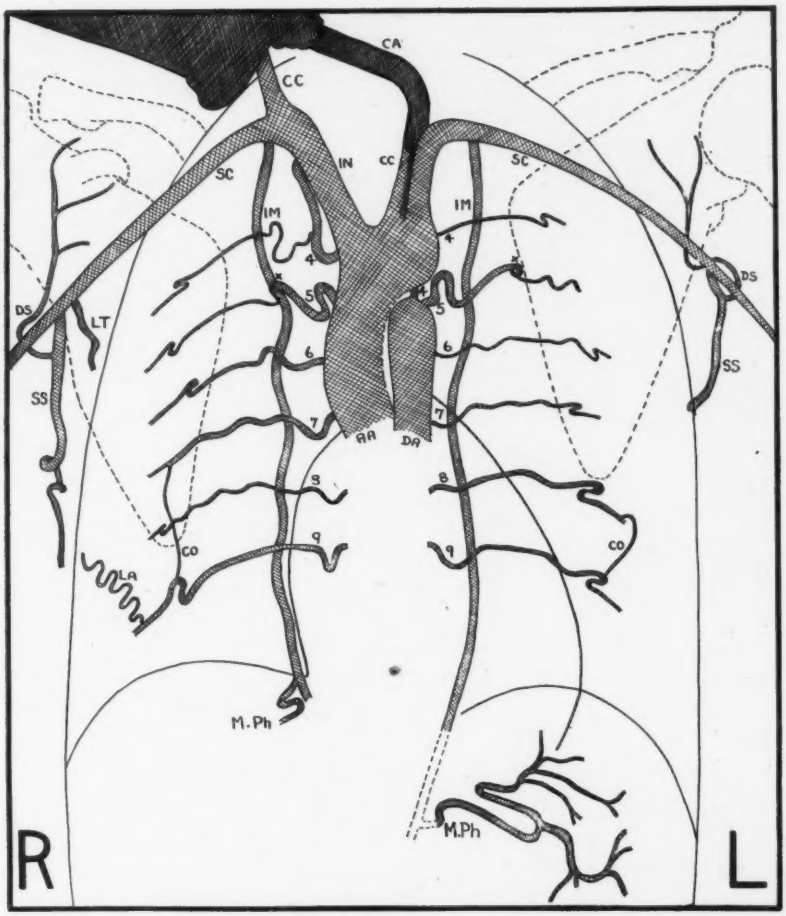


FIG. 8.—*Tracing from Fig.* 7.

CA—Injection Canula.

ARTERIES.

AA—Ascending aorta.
IN—Innominate.
SC—Subclavian.
LT—Long thoracic.
DS—Dorsalis scapulæ.
LA—Anastomosis of subscapular with aortic intercostals.
4, 5, 6, etc.—Aortic intercostals.
DA—Descending aorta.
CC—Common carotid.
IM—Internal mammary.
SS—Subscapular.
M.Ph.—Musculo-phrenic forming diaphragmatic anastomoses.
CO—Communicating branches between intercostals.
XX—The points at which the second aortic intercostals (supplying the fifth spaces) notch the ribs.

FIG. 9.—*Barium injection: Radiogram in the left anterior (II) oblique position, at the end of injection.* The coarctation is clearly seen and is crossed by two tortuous and enlarged aortic intercostals passing almost vertically upwards to their anastomosis with the superior intercostal. The extensive diaphragmatic anastomosis is seen.

FIG. 10.—*Barium injection : Radiogram of the aorta in the left anterior (II) oblique position.* The coarctation is shown and appears to be complete. The aortic valve and the sinuses of Valsalva are seen. The valve is obviously competent.

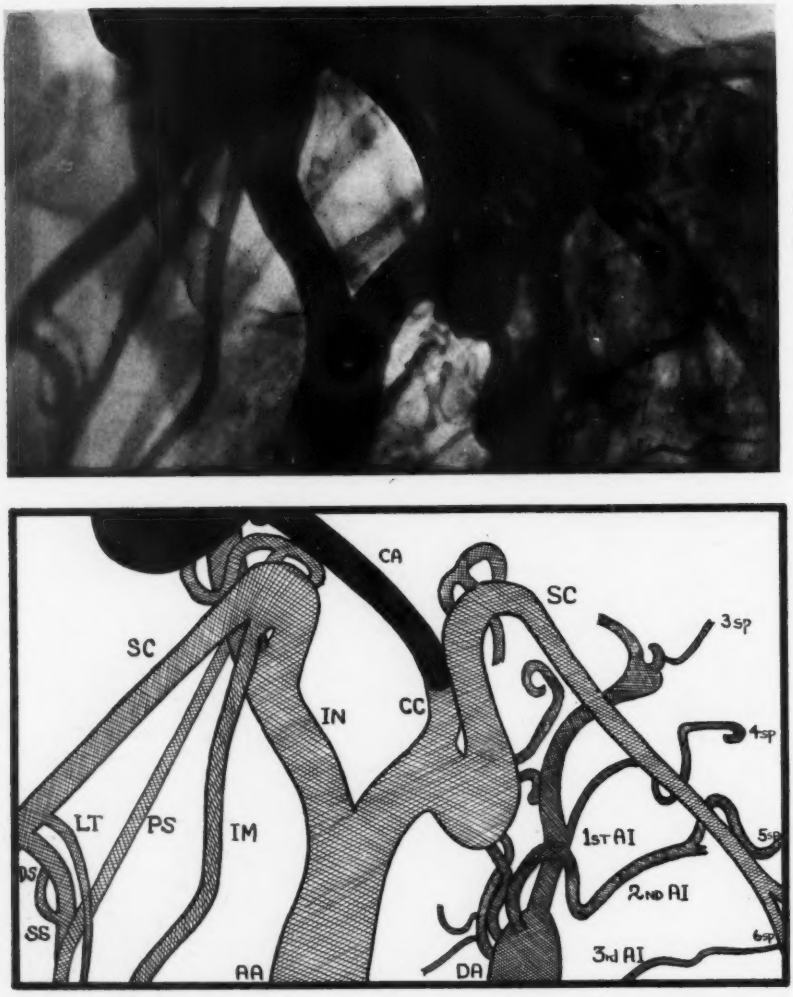


FIG. 11.—*Radiogram in a left oblique position, showing the coarctation.*

Key.—The arteries arising below the coarctation are represented in a darker shade.

CA—Injection Canula.

LT—Long thoracic. DS—Dorsalis scapulæ. IN—Innominate.
PS—Posterior scapular. SS—Subscapular. CC—Common carotid.
IM—Internal mammary. SC—Subclavian. AA—Ascending aorta. DA—Descending aorta.
1st AI, 2nd AI, etc.—first and second left aortic intercostals, etc.
3sp, 4sp, etc.—branches of left aortic intercostals to third and fourth spaces, etc.

The left subclavian forms a continuation of the aortic arch. The first left aortic intercostal (1st AI) runs almost vertically upwards, and gives off branches to the fourth and third intercostal spaces (4sp and 3sp); its main trunk anastomoses with the superior intercostal. The second and third left aortic intercostals (2nd AI, 3rd AI) are visible. The origins of the right aortic intercostals are hidden by the descending aorta (DA).

FIG. 12.—*Barium injection: Radiogram of the abdominal vessels.* The complete injection of the abdominal vessels is shown. As the barium was all injected above the coarctation these must have filled by the collateral channels. The well-known anastomosis between the superior and deep epigastric arteries was absent in this case.

Discussion

Clinically, the collateral circulation is revealed by palpable dilated arteries on the back or front of the chest, and by the late systolic murmur sometimes audible over these vessels; radiographically, its important manifestation is notching of the ribs by the enlarged and tortuous intercostal arteries. These features merit further consideration.

The Anastomotic Pattern

The blood has a choice of routes whereby to reach the aorta beyond the coarctation, and it would be interesting to know whether the circulatory efficiency in different cases is related to the anastomotic pattern.

In Table I we have enumerated, and in Fig. 13 we have endeavoured to depict diagrammatically, the various collateral channels which may be of importance.

TABLE I

Collateral Channels in Coarctation of the Aorta

1. The Scapular and Cervical Anastomoses:

The following arteries form a network around the scapula and in the cervical region:

(a) Suprascapular and transversalis colli (from thyroid axis).
(b) Posterior scapular and superficial cervical (from transversalis colli).
(c) Long thoracic and subscapular with its dorsalis scapulæ branch (from axillary artery).

From this network descending branches anastomose with the lateral and dorsal branches of the aortic intercostals.

2. The Internal Mammary Anastomoses:

(a) Superior epigastric → deep epigastric branch of external iliac.
(b) Musculo-phrenic → phrenic branches of thoracic and abdominal aorta.
(c) Mediastinal branches → mediastinal branches of aorta.
(d) Anterior intercostals → terminal branches of aortic intercostals.

3. The Intercostal Anastomoses:

(a) The terminal branches → the intercostal branches of the internal mammary.
(b) The lateral branches → the subscapular and long thoracic.
(c) The dorsal branches → the posterior scapular.
(d) The first and second intercostals (arising from the subclavian by the superior intercostal) → the upper aortic intercostals.
(e) Each intercostal → those above and below.

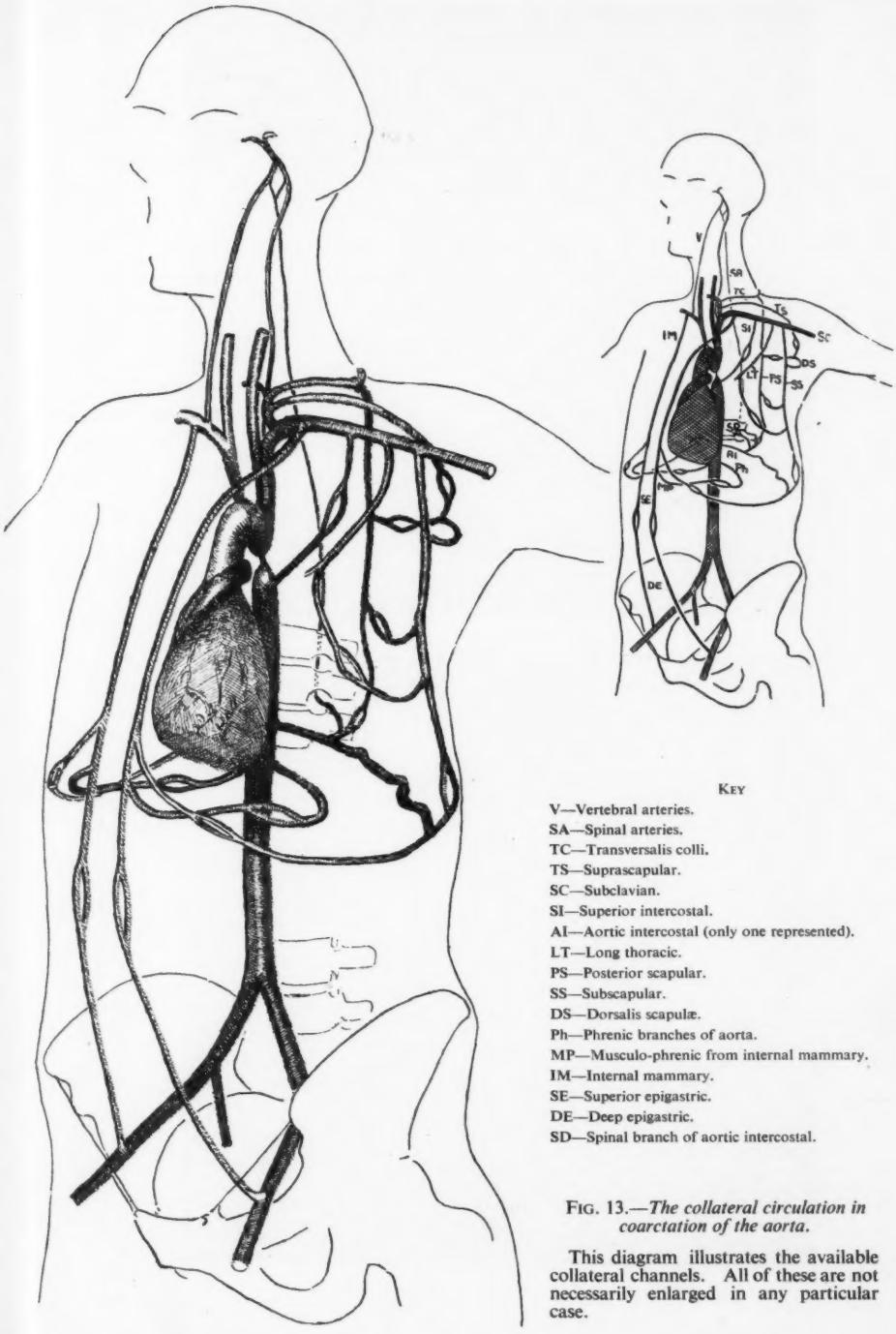
4. The Spinal Anastomoses:

The vertebral artery, arising from the first part of the subclavian, reinforces the spinal arteries in which the blood flows downwards to reach the spinal branches of the aortic intercostals. These pass through each intervertebral foramen. There are also branches from the inferior thyroid which pass through the intervertebral foramina in the neck to join the spinal arteries.

In our case the main anastomoses were :

(a) By the scapular and cervical branches of the subclavian and axillary arteries to the lateral and dorsal branches of the aortic intercostals, forming a network around the scapula and in the neck.

(b) By the musculo-phrenic branch of the internal mammary to the



KEY

V—Vertebral arteries.
SA—Spinal arteries.
TC—Transversalis colli.
TS—Suprascapular.
SC—Subclavian.
SI—Superior intercostal.
AI—Aortic intercostal (only one represented).
LT—Long thoracic.
PS—Posterior scapular.
SS—Subscapular.
DS—Dorsalis scapulæ.
Ph—Phrenic branches of aorta.
MP—Musculo-phrenic from internal mammary.
IM—Internal mammary.
SE—Superior epigastric.
DE—Deep epigastric.
SD—Spinal branch of aortic intercostal.

FIG. 13.—*The collateral circulation in coarctation of the aorta.*

This diagram illustrates the available collateral channels. All of these are not necessarily enlarged in any particular case.

FIG. 14.

FIG. 15.

Rib notching. The conspicuous notch on the right ninth rib is shown before (Fig. 14) and after (Fig. 15) injection of the intercostal artery. The outer notch has a characteristic a symmetrical shape, with a gradual slope on one side and a steep slope on the other; the way in which the arterial loop produces a notch of this shape is seen. The inner notch does not show this characteristic shape. See text, p. 222.

inferior phrenic branches of the abdominal aorta and the smaller superior phrenic branches of the thoracic aorta, forming a network below and above the diaphragm.

(c) By the upper two intercostals, from the superior intercostal, to the upper aortic intercostals.

The importance of the anastomosis between the superior and deep epigastric has been stressed in most accounts, possibly because it is easily palpable. In our case (Fig. 12) it was insignificant. Likewise the anastomosis between the anterior intercostals (arising from the internal mammary) and the terminal branches of the aortic intercostals was poorly developed, the great enlargement of the internal mammary being due chiefly to the extensive diaphragmatic anastomosis. Unfortunately, at the time these observations were made we did not realize the importance of the spinal anastomosis (Table I and Fig. 13), and consequently no attempt was made to study it.

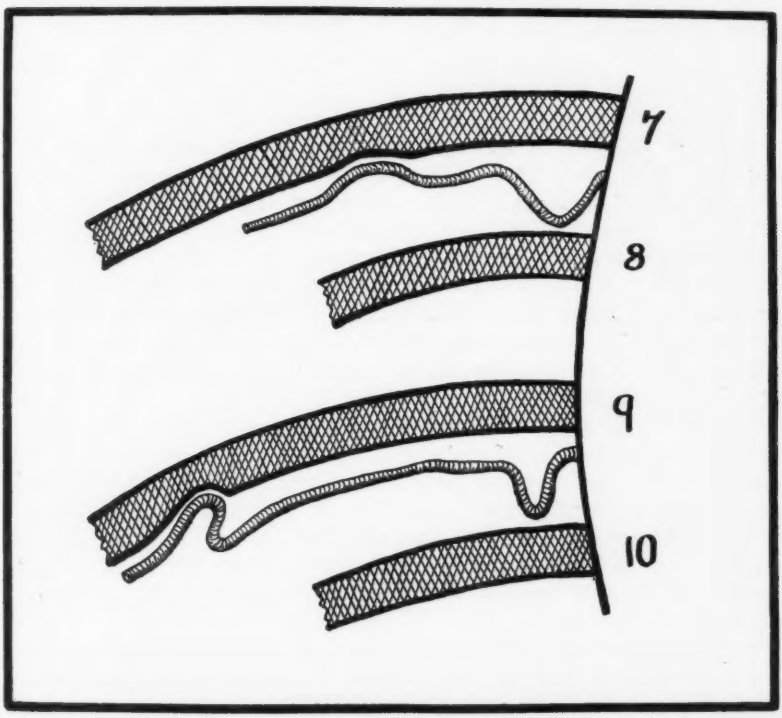


FIG. 16.—*The formation of rib notches.* The two intercostal arteries illustrated are drawn from the seventh and ninth right intercostals shown on Fig. 7. The seventh intercostal forms a smooth, shallow, symmetrical erosion on the seventh rib, but the greater tortuosity of the ninth intercostal produces a notch of characteristic shape. This type of notch can be seen on several ribs in Fig. 7. The drawing also illustrates how the intercostals may be very tortuous near the necks of the ribs without notching them, since here the arteries are some distance from the lower margins of the ribs (see text, p. 222).

Rib Notching

(a) *The Formation of Notches.* It was suggested by Arendt (quoted by Wolke, 1937) that rib notches in coarctation were of two types: shallow notches due to the tortuosity of the arteries, and deeper, punched-out notches due to small aneurysms on the intercostal arteries. Wolke (1937) studied radiographically a single aortic intercostal injected with iodized oil and arrived at the conclusion that it was unnecessary to invoke aneurysm formation, as the tortuosity of the artery was sufficient to account for both types of notch.

Our observations confirm those of Wolke. Fig. 14 and 15 (p. 220) illustrate how an arterial loop may form a notch, and in Fig. 16 we have represented diagrammatically the formation of shallow and deep notches by the different degrees of tortuosity of the intercostal artery. From Fig. 14 and 15 it is clear that the deep notches have a very characteristic shape; they are asymmetrical, with a gradual slope on one side and a steep slope on the other. This shape is determined by the form of the arterial loop, which is remarkably constant; several such loops are visible in Fig. 7.

(b) *The Distribution of Notching.* The distribution of notching is interesting. The notches in the present case were on the fourth, fifth, sixth, seventh, and ninth ribs on the right and on the fourth, fifth, sixth, eighth, and ninth ribs on the left (Fig. 17). We have studied the distribution of the notching in the radiograms of the other twelve cases in our series. This is tabulated in Table II, (p. 226) illustrated in Fig. 18 (p. 224), and represented graphically in Fig. 19.

The data enumerated indicate that notching is commonly confined to the ribs between the third and the ninth. It is absent in the first two ribs because the upper two intercostal arteries arise from the subclavian by the superior intercostal ; unlike the other intercostals, they arise above the coarctation. Near their origin they give off large branches, which anastomose with the upper aortic intercostals; after this they play no further part in the collateral circulation and do not become tortuous.

The aortic intercostals supply the intercostal spaces from the third downwards. They become tortuous because a large amount of blood enters their dorsal and lateral branches from the scapular network. The number of aortic intercostals that enlarge depends upon the site of termination of the subscapular, posterior scapular, and other descending branches of the scapular network. The shorter collateral channels to the upper aortic intercostals naturally enlarge first, and this is probably the reason why notching seldom extends below the ninth rib.

The notches are always confined to the posterior and lateral portions of the ribs; the anterior portions are never involved. This is probably due to the fact that the main blood flow enters the intercostals by their lateral and dorsal branches, whereas the anterior intercostals, arising from the internal mammary, are comparatively unimportant.

Sometimes, as in the present case, a double series of notches occurs (Fig. 17). When this is so, the inner notches are almost invariably confined to the third, fourth and fifth ribs (Table II and Fig. 18 and 19). The third, fourth, and fifth

intercostal spaces are the first to be supplied by the aortic intercostals. In the present case the first aortic intercostal supplied the third and fourth spaces; it was greatly enlarged, and its main trunk anastomosed with the superior intercostal (Fig. 11). The second aortic intercostal supplied the fifth space.

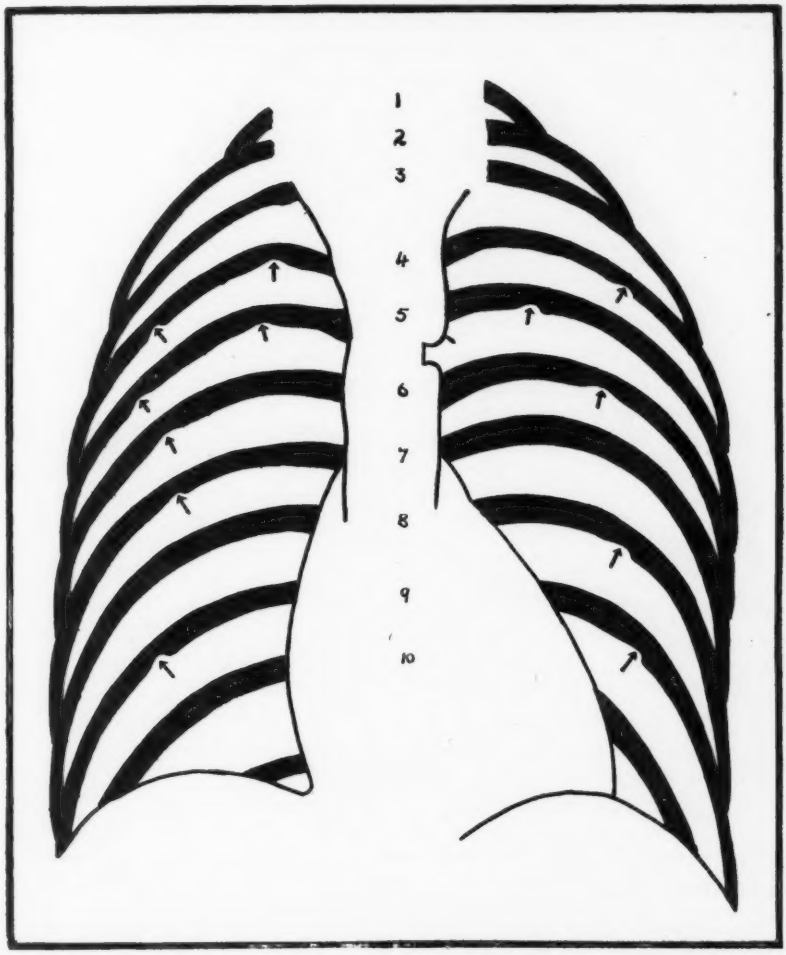


FIG. 17.—*The position of rib notches.* This diagram is taken from Fig. 6 and illustrates the position of the definite notches in this case.

It was also considerably enlarged, but on both sides it diminished suddenly in size about two inches from the aorta. This occurred because at this point the artery gave off a large branch, which turned upwards towards the superior intercostal. At its origin this branch notched the fifth rib on each side (Fig. 7 and 8); these notches are represented in Fig. 17 and are examples of the

inner group of notches depicted in Fig. 18. The anastomosis between the upper aortic intercostals and the superior intercostal therefore appears to be responsible for the production of the inner group of notches. This accounts

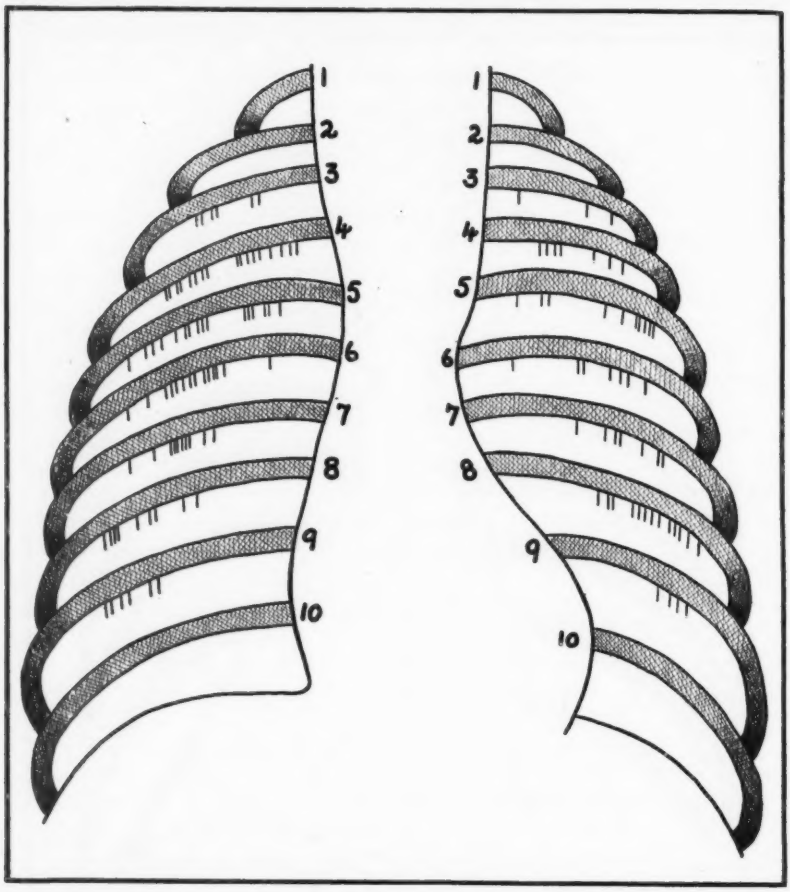


FIG. 18.—*The distribution of rib notches in thirteen cases of coarctation of the aorta.* The position of the definite notches seen in A.-P. radiograms of thirteen cases have been represented on a single diagram in order to show their positions on each rib and to indicate the ribs most frequently notched. The notches may be separated into an inner group on the third to the sixth ribs and an outer group on the third to the ninth ribs.

for the position of the notches near the spine and for the fact that they occur only on the third, fourth, and fifth ribs.

The outer group of notches is due to the anastomosis of the aortic intercostals with the descending branches of the scapular network and has a different distribution. These notches are situated more laterally, they extend as low as the ninth rib, and are most frequent from the fifth to the eighth ribs (Fig. 19).

The distribution of the notches in the present case is illustrated in Fig. 17 and is typical. It is striking that the outer notches tend to lie vertically under one another (Fig. 17) and in a fairly constant position (Fig. 18). Notching is due to tortuosity of the arteries, and tortuosity is most likely to occur midway between two fixed points on the artery. In searching for such fixed points one naturally thought of the nutrient branches of the ribs, but these are inconstant in position. The origins of the dorsal and lateral branches of the intercostals,

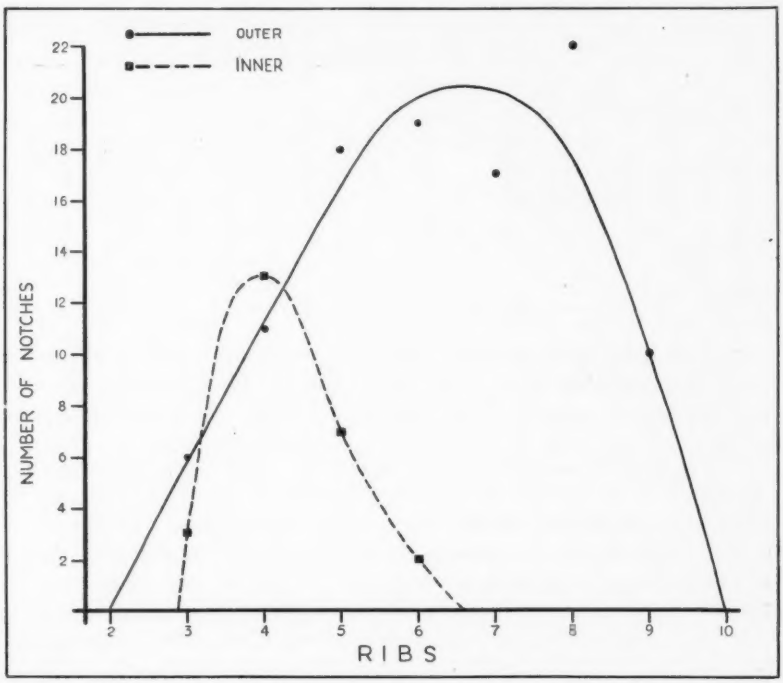


FIG. 19.—*Graph illustrating the distribution of rib notching in coarctation of the aorta.* The distribution of rib notching illustrated in Fig. 18 has been represented graphically. The curves illustrate how the outer notches occur most frequently on the fifth, sixth, seventh, and eighth ribs, whereas the inner notches are commonest on the fourth rib.

on the other hand, are fairly constant in position, and, because these branches pierce the fascial and muscular planes of the chest wall, they anchor the intercostal arteries at these points. The position of the outer notches supports the hypothesis that tortuosity first develops midway between these two points. The communications between the seventh intercostal and the loop on the ninth right intercostal and between the eighth left intercostal and the loop on the corresponding ninth intercostal (Fig. 7, 8, and 15) are interesting, for they may account for the position of some arterial loops.

Another fixed point is the origin of the intercostal from the aorta. Between this point and the origin of the dorsal branch considerable tortuosity may

develop (Fig. 7 and 8). This was illustrated diagrammatically in Fig. 16. It is, however, uncommon for notching to occur in this situation, for here the intercostal arteries are not in such close relation to the ribs.

TABLE II

DISTRIBUTION OF RIB NOTCHING IN THIRTEEN CASES OF COARCTATION OF THE AORTA

Rib	Right			Left			Total Outer	Total Inner	Complete Total
	Outer	Inner	Total	Outer	Inner	Total			
1	—	—	—	—	—	—	—	—	—
2	—	—	—	—	—	—	—	—	—
3	4	2	6	2	1	3	6	3	9
4	8	9	17	3	4	7	11	13	24
5	10	6	16	8	3	11	18	9	27
6	13	1	14	6	1	7	19	2	21
7	10	—	10	7	—	7	17	—	17
8	9	—	9	13	—	13	22	—	22
9	6	—	6	4	—	4	10	—	10
10	—	—	—	—	—	—	—	—	—
11	—	—	—	—	—	—	—	—	—
12	—	—	—	—	—	—	—	—	—
Totals	60	18	78	43	9	52	103	27	130

Rib notching may occur quite early in life. It was well developed in our youngest patient, a boy of 11, and Brown (1939) states that it has been reported at the age of 6.

Laubry (1937) has reported notching of the ribs in conditions other than coarctation of the aorta. He described six cases of aortic incompetence and of hypertension in which notches were seen. These differed from the notches in coarctation, for they occurred as a rule in elderly subjects, they were shallow, and limited to a few of the lower ribs, and were usually near the spine. They must be extremely uncommon, and their existence does little to diminish the diagnostic value of the discovery of rib notching in coarctation of the aorta.

Pain

Our patient's principal complaint was pain on exertion, in the scapular region; and pain was the presenting symptom in three other cases in our series. In one of these the patient had first noticed pain in the lower interscapular region at the age of 17. This persisted, and, at the age of 21, led him to consult an osteopath, who treated him unsuccessfully for eighteen months by manipulation of the spine. In the other two cases the pain was in the right arm and around the left costal margin respectively.

In two of the four cases reported by King (1926) pain was a prominent symptom. One of his patients, a man of 35, complained of pain on exertion in the left side of the chest; a year later a similar pain developed in the right chest. In King's other patient, a man of 58, the pain was in the left supraclavicular fossa and left shoulder, and corresponded in position to an area of arterial pulsation. It occurred only on exertion and for a considerable period it was the only symptom.

These cases illustrate that pain may occur in several situations, but it is always confined to the upper part of the body. It is in this area that the collateral circulation is developed, and in one of King's cases the pain was localized to an area of arterial pulsation.

Enlarged collateral channels might produce pain in several ways. When the pain is in the back it is possible that it may be due to erosion of the ribs, but the frequency with which rib notching occurs without pain throws doubt on this explanation. Pain in the arm, or around the costal margin, is more suggestive of root or nerve pressure, which might be due to the enlarged anastomotic artery passing through the intervertebral foramen (Fig. 13). The relation of the pain to exertion could be explained by the increased blood flow through the tortuous and pulsating collateral channels, and the differing sites of pain may be related to differences in the anastomotic pattern.

These various considerations suggest that the enlarged pulsating vessels of the collateral circulation are instrumental in the production of this symptom, and that in some instances the pain may be due to nerve pressure or to the erosion of bone.

Summary

1. The post-mortem findings in a patient with coarctation of the aorta who died from subarachnoid hæmorrhage due to a ruptured cerebral aneurysm have been described.

2. As the coarctation had been diagnosed and the condition fully investigated two years previously, it was possible to compare the clinical and post-mortem findings.

3. Prior to autopsy the cadaver was injected with barium paste and the arterial anastomoses studied radiographically.

4. The anatomy of the collateral circulation has been described. The factors that determine the distribution of rib notching and some possible causes of pain in this condition have been discussed.

We are grateful to Sir John Stopford for his help and interest in this work; to Mr. F. S. A. Doran, who kindly drew for us Fig. 13; to our colleague, Dr. E. Duff Gray, for the facilities afforded to us in his department, and to Dr. S. Nowell for his valuable help in the radiography of the cadaver.

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CHANGES IN RENAL FUNCTION AND PERSISTENCE OF THE MURMUR AFTER LIGATURE OF A PATENT DUCTUS ARTERIOSUS

BY

GEOFFREY BOURNE

Received July 24, 1941

When first seen the patient here referred to was suffering from infection of a patent ductus arteriosus by *Streptococcus viridans*. Ligature of the ductus and treatment by sulphapyridine effected a cure. Full clinical and operative details are described, together with a second case of patent ductus arteriosus infected by *B. influenzæ* and cured by ligation and chemotherapy, in a paper by Bourne, Keele, and Tubbs (1941).

The reason for the present publication is the existence of the following two points of interest, additional to the fact of cure, points which seem to deserve separate and full description :

(I) As a result of obliteration of the ductus there resulted a temporary but striking and prolonged impairment of renal function, associated with a marked increase of the diastolic blood pressure ;

(II) In spite of obliteration of the ductus the typical murmur persisted for some months.

The patient, a girl of 19, had been in good health until four months previous to her admission to St. Bartholomew's Hospital. She had suffered during this time from headache, lassitude, and undue sweating at night. Fever was present throughout this period. No peripheral or pulmonary embolic symptoms were reported or found. The heart was enlarged, especially to the left. The typical systolic thrill and murmur of patent ductus arteriosus were present. The blood pressure was 132/40. The pulmonary conus was seen radiographically to be enlarged. Blood culture proved on several occasions the presence of *Streptococcus viridans*. The growth became progressively less profuse with sulphapyridine therapy, and before operation the blood was sterile.

Since the ligation of the ductus, and for a period of nine months, she has remained perfectly well.

The following sentences are quoted from Mr. Tubbs' account of the surgical procedure, with slight abbreviation and modification. The ductus arteriosus was approached through the left pleural cavity, which was opened through the second left intercostal space, the skin incision extending from the mid-line in front to the anterior axillary line. The second and third costal cartilages were

divided close to the sternum, and excellent exposure was obtained by separating the ribs with a " rib-spreader ". The internal mammary vessels were divided between ligatures so that full use of the medial end of the incision might be obtained. The mediastinal pleura was incised parallel and posterior to the phrenic nerve, which was then retracted anteriorly. No difficulty was experienced in identifying the patent ductus. The cellular tissue under the arch of the aorta was gently swept from the anterior surface of the ductus with a pledget of gauze held in a long hæmostatic clamp. Keeping immediately against the wall of the ductus, the adjacent tissues were readily separated by blunt dissection, except from the postero-medial surface, where a plane of cleavage was found by tactile means and without visualization. This was done by insinuating the left index finger into the cleft between the aortic arch and the bifurcation of the pulmonary artery, anterior to the ductus. None but a trivial amount of hæmorrhage was encountered. The ductus was three-quarters of an inch in length and rather more then half an inch in diameter. It was obliterated by two doubled No. 5 tubular silk ligatures.

Renal Functions and Blood Pressure Changes

Previous to operation the average blood pressure reading was 130/40, and the urine, which contained no albumin or cells or casts, was sterile on culture. After the operation, and on the same day, the systolic blood pressure fell to 112, but in five hours rose to 130, the corresponding diastolic figures being 84 and 94. On the following day the figures were at first 136/116, and later 116/100. On the second and third days there was little change, but on the fourth day after operation the figures were 154/122 and 150/120. From this point the pressures slowly fell, reaction of 140/105 on the eleventh day. The systolic pressure then became reduced during the next week to between 130 and 140, where it remained, but the diastolic continued in the neighbourhood of 94, and not until six weeks after operation did it fall to the final permanent figure of 74 to 78.

During the whole of this post-operative phase no abnormal cells, no casts, and no albumin were found in the urine, but there was a gross diminution in renal efficiency. Eight days following operation the urea content of the blood was 62 mg. per 100 c.c., and fifteen days afterwards this had increased to 72 mg. per 100 c.c. On this day the standard urea clearance was 21·9 c.c., or 29·2 per cent of the average normal for the first hour, and 15·2 c.c., or 28 per cent, for the second hour. No urinary abnormality was found even at this stage. The urea clearance figures then slowly increased, so that on the twenty-third day after operation the figures were 53·5 per cent of the average normal for the first hour and 40·8 per cent for the second.

Two months later the respective figures were 66·6 per cent and 60·3 per cent. This renal failure was closely parallel in degree with the increase and subsequent return to normal of the diastolic blood pressure, and it had the characteristics of pure glomerular failure.

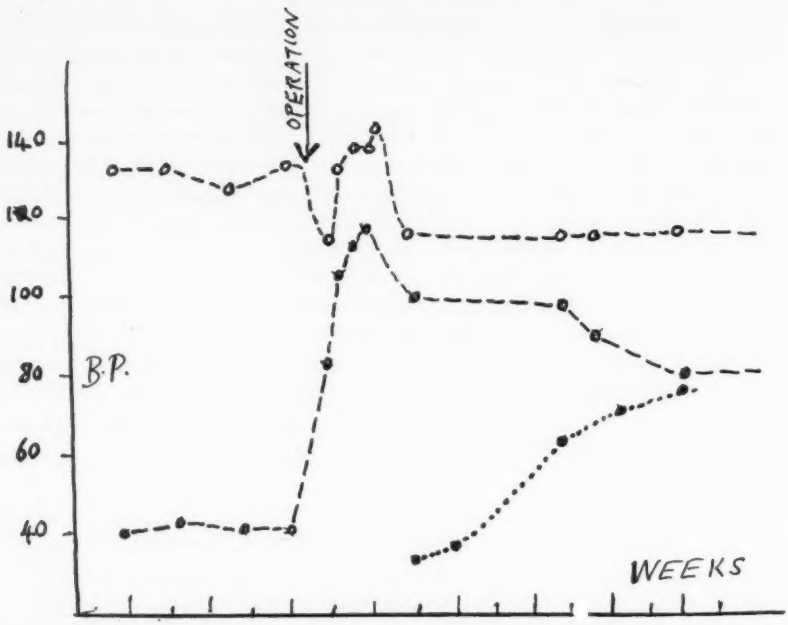

FIG. 1.—Changes in the blood pressure and renal function after ligation of a patent ductus arteriosus.

Divided lines : blood pressure, systolic and diastolic.
Dotted line : renal function, urea clearance, percentage of average normal.

The following hypothesis is offered to explain why the blood pressure and renal changes may have occurred.

Before operation the state of the cardiovascular system was as follows : The left ventricle was hypertrophied. The output per beat from this chamber was greater than normal. The total blood volume was larger than normal (Gross, 1939). The pulse curve was that of aortic incompetence, in which the peripheral arterioles were in a condition of more or less permanent partial relaxation. This cardiovascular state was normal for the patient, having been present for the whole of her nineteen years of life.

After operation the volume of the blood would have adjusted itself within a few days, and this can hardly be considered as a factor of significance. The left ventricular hypertrophy would remain for a number of weeks but might then disappear ; for when hypertrophy of the heart is produced in rabbits by experimental arterio-venous aneurysm it has been observed to disappear when the leak is closed again (personal communication, Dr. A. N. Drury). This has also been observed clinically in cases of arterio-venous aneurysm (Hitzig and Master, 1935). But during these weeks the ventricular output per beat might be expected to be increased above normal. This alone, however, could hardly be responsible for the renal and blood pressure changes.

It would seem that attention should therefore be directed to the state of

the peripheral arterioles. For the whole of the patient's life the nervous tone of the peripheral vessels had been adjusted for the arterial leak from the patent ductus ; and also stabilized as regards their neuro-muscular mechanism to react to the type of pulse curve found characteristically in aortic regurgitation. The vessel would thus have been in that state of permanent partial dilatation associated with a lowered diastolic blood pressure.

It is suggested that the effect of the increased output per beat upon this hitherto constantly relaxed vascular bed could produce a reflex over-reaction. This abnormally hypertonic state of the arterioles might be expected to continue until the vasomotor nervous system had readjusted itself to the new conditions, or until the hypertrophy of the left ventricle had become sufficiently resolved. The period of readjustment might persist for some weeks.

If this vascular spasm involved the glomerular as well as the systemic arterioles, a single explanation is available for the abnormally high diastolic pressure following operation, for the coexisting diminution in renal function, and for the fact that the urine at no time showed any cells, casts, or albumin.

It may be objected that the renal changes had no association with the tying of the ductus and the subsequent blood pressure variations but were the direct result of the infection ; and that when the infection was cured they naturally disappeared. In answer to this are the facts that although the blood urea was at one time 72 mg. per 100 c.c., at no time were red blood cells, casts, or albumin found in the urine, as is usually the case when kidney damage during endocarditis or septicæmia is extensive ; and no embolic signs were present elsewhere. Nor would recovery in such a case have been complete in so short a period. Finally, the relation between the renal and blood pressure changes is obviously a close one.

After ligature of the ductus in Case I (Bourne *et al.*, 1941) the diastolic pressure rose from 60 to 120, but later fell to the region of 80.

Similar elevation of diastolic pressure is characteristic of closure of arterio-venous aneurysms, both in man and in experimental animals, as Holman (1940) has pointed out in a full review of the subject.

Persistence of the Murmur

The murmur of patent ductus arteriosus is typically one that fills systole and is carried on into diastole. During this period the pressure in the aorta is at first systolic, and then, in proportion to the size of the arterial leak, it falls to a lower diastolic level than normal. The murmur is loudest at those periods of the cardiac cycle when the flow of blood from aorta to pulmonary artery is most rapid. This provides the classical explanation that the murmur is directly caused by this flow. In the case here described the murmur before operation was classical, and the aortic leak was large, but after ligature of the ductus the murmur persisted, although the leak was stopped. The blood pressure was completely changed by the operation, the figures before operation being 132/40, and after operation 136/116 at first and 130/80 finally. Two doubled tubular silk ligatures were used, and there is no reason to doubt that

they were effective. The persistence of the murmur was verified by four independent observers. It remained definite for three months, and six months later was far less loud, and its diastolic part is now (after nine months) nearly inaudible. Touroff (1940) noticed the same persistence of the murmur in two of his cases after ligature, but the length of its duration was unstated. In one of his cases the ductus was cut between two ligatures, thus disposing of a possible criticism suggesting that the ligation had been imperfect. It is clear that in all these cases the classical murmur was not caused in the manner usually suspected. It is therefore possible that it is never so caused ; its mode of production must therefore remain a matter for conjecture. Ligature of the patent ductus did, however, result in disappearance of the murmur in our Case 1 (Bourne *et al.*, 1941). Moreover Gross (1939), after the ligature of four non-infected cases, noted persistence of the double murmur, but with diminished loudness, in two cases, persistence of a soft systolic murmur in one case, and disappearance of the murmur in one case. At operation a faint systolic thrill could be palpated in the writer's case in the pulmonary conus after applying the first ligature, but after the second this was abolished.

Summary

A case of patent ductus arteriosus is described, in which ligature of the ductus

(1) caused a marked increase in the diastolic pressure, associated with great impairment of renal function, and

(2) did not result in disappearance of the classical murmur.

The hitherto undescribed changes in renal function and the persistence of the murmur, after ligature, are reported so that in future cases these points may be further investigated. Careful renal function studies should be done before as well as after operation in such cases.

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INVERSION OF THE T WAVES IN LEAD II CAUSED BY A VARIATION IN POSITION OF THE HEART

BY

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Received September 9, 1941

Only in late years has there begun to be adequate recognition of the wide variations of the normal human electrocardiogram. As has so often happened in medical science, diseased conditions and abnormal findings have attracted so much attention that a patient study of the normal subject and his physiological variations has been left far behind. This is particularly true in clinical electrocardiography where we still depend for our normal criteria on a relatively small number of records.

Leimdörfer (1935) reported the finding of a lowering, to an isoelectric or diphasic position, of the T waves of lead II in certain subjects on changing their body position from supine recumbency to standing.* This alteration of the T waves he ascribed to heart disease, not otherwise apparent; for this viewpoint, however, he had no adequate evidence. That same year, two of us (P.D.W. and A.G., 1935) published a paper in which we called attention to the occasional occurrence of inversion of the T waves in lead II in persons without heart disease and in fact sometimes without any obvious disease at all. Several of these were young people of delicate build and subject to neurocirculatory asthenia. We stated that the reason for T wave inversion in these cases was unknown but that " the importance of this finding is that it points to the need of caution in the diagnosis of serious heart disease when inversion of the T wave in lead II is the only abnormal finding." In these cases the T waves in lead III were always more deeply inverted than those in lead II and " remained inverted in most instances (16 out of 23) after the T wave in lead II had ceased to be inverted."

As time went on after our own initial report we became cognizant of at least one of the factors which was responsible for the inversion of the T waves

* The records taken in the standing position were made with the breath held in full expiration with the express purpose of neutralizing the effect of the change in the position of the heart; it is our experience that with ordinary breathing in the upright position the T waves would often have been inverted in lead II, rather than merely isoelectric or diphasic, and that change in position of the heart with expiration alone may be insufficient to neutralize the effect of change in body position.

in leads II and III. We observed that the position of the body was of major importance and that a change from the upright position (seated) to recumbency (supine) often altered the T waves so that they became upright in lead II and less inverted, flat, or even upright in lead III. In 1938 we reported our findings in brief before the American Society for Clinical Investigation (by title and abstract) and before the New England Heart Association. We stated then that the electrocardiograms usually became normal with changes of position and after exercise. Electrocardiographic abnormalities reversible with changes in posture (sitting to supine) were often seen as transient phenomena associated with respiratory infections. The patients were mostly young adults of a nervous temperament and of asthenic habitus. Myocardial function, electrolyte balance, and heart position in relation to various body positions were studied and only the latter found to have a bearing on the electrocardiographic changes.

A variation similar to what we had encountered was reported by Åkesson (1936) and ascribed to orthostatic coronary insufficiency. He observed, among other things, that the greater the increase in pulse rate and the greater the fall in systolic blood pressure, when the subject stood erect, the greater was the decrease in the height of T in lead III. Åkesson, however, failed to distinguish between the T wave changes that occur immediately on assuming the upright position and those that occur as a result of the pooling of blood in the lower portions of the body. We too have observed significant changes in the T waves of the electrocardiogram associated with orthostatic hypotension and tachycardia, and believe they are due to alterations in sympathetic and vagal tonus and perhaps to a decrease in coronary blood flow. However, in the observations which we report below, this mechanism did not play a significant rôle.

Sigler (1938) reported the occasional occurrence of electrocardiographic alterations with change in body posture, including in a few cases inversion of the T waves in lead II when the subject assumed the erect position, which he ascribed to " a change in contact of the adjacent conducting media with different portions of the heart on alteration of body posture, producing variation in conduction," and not to heart disease as Leimdörfer had done.

Two years ago an important observation came to our attention in helping to clear up the mystery of the inversion of the T waves in lead II in certain normal subjects. It was already evident that posture was important but just how a change in posture acted was not wholly clear. We now found that without a change in posture the typical changes in the T waves described above could sometimes be produced by deep inspiration and sometimes abolished by deep expiration. In other words, the height of the diaphragm is almost, if not quite, as important a factor as is body position, thus pointing to heart position in relation to other parts of the body as of prime significance. Rotation of the heart on its anteroposterior axis is probably a minor cause of the change, since often there is but little change in the angle of the electrical axis of the heart in the classical frontal plane of the body as measured from the QRS waves by the Einthoven formula or triangle. Rotation of the heart on its longitudinal axis is probably a more important factor in influencing the T waves with change in the heart's position. Nevertheless, it should be

recognized that the electrical axis of the T waves and of the QRS complexes are distinguishable and that an alteration or shift in the QRS axis need not be associated with a comparable shift in the T axis and vice versa.

Electrocardiograms illustrating the effect of posture and of respiration on the T waves in leads II and III were published by two of us (P.D.W. and A.G.) in our new book on electrocardiography (1941). Records of one case (Fig. 15 and 16 of the book) are reproduced herewith as Fig. 1. Other typical illus-

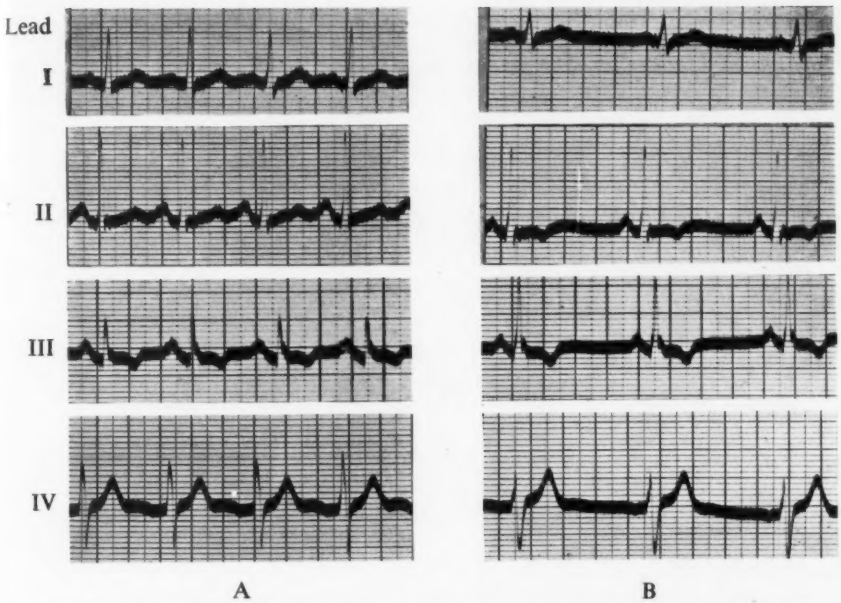

FIG. 1.—Electrocardiographic changes with respiration in a healthy young man of stocky build. (A) During quiet respiration. (B) In full inspiration.

Note that here there is a distinct shift in the angle of the electrical axis of the QRS waves to the right as well as the inversion of the T waves in lead II upon lowering the diaphragm and making the heart much more vertical in its position. There may or may not, however, be much of any change in the QRS waves with change in the T waves.

trations are shown in Fig. 2, 3, 4, and 5 of the present paper. It should be noted that changes in the S–T junctions and segments and in the T waves in leads I and IV are relatively slight and unimportant in comparison with the changes in the late phases of the T waves in leads II and III.

Recently Scherf and Weissberg (1941) have reviewed the subject and have arrived at the same conclusions as ourselves; they pointed out that the T waves in lead II may be simply lowered rather than inverted, and emphasized particularly the inversion of T in lead III and its change in 35 normal adults. They concluded that their " observations speak against the assumption that cardiac damage or anoxia of the heart muscle can be the factor responsible for the electrocardiographic alterations. They confirm the conception that the

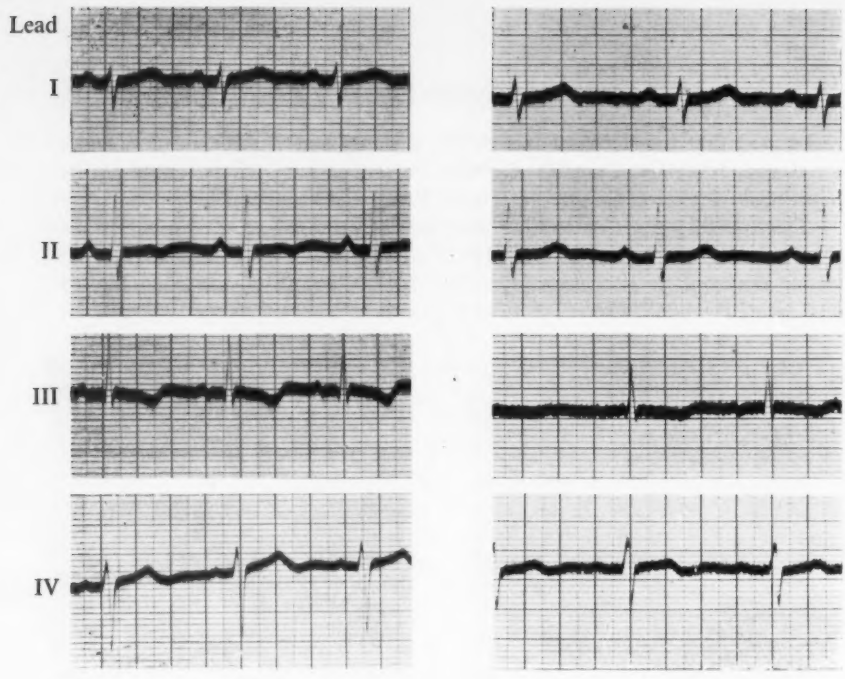

FIG. 2.—Electrocardiographic changes upon change in position from sitting to supine in the case of a young girl, 15 years old and of average build, with no clear evidence of heart disease but of nervous type. (A) Sitting. (B) Recumbent.

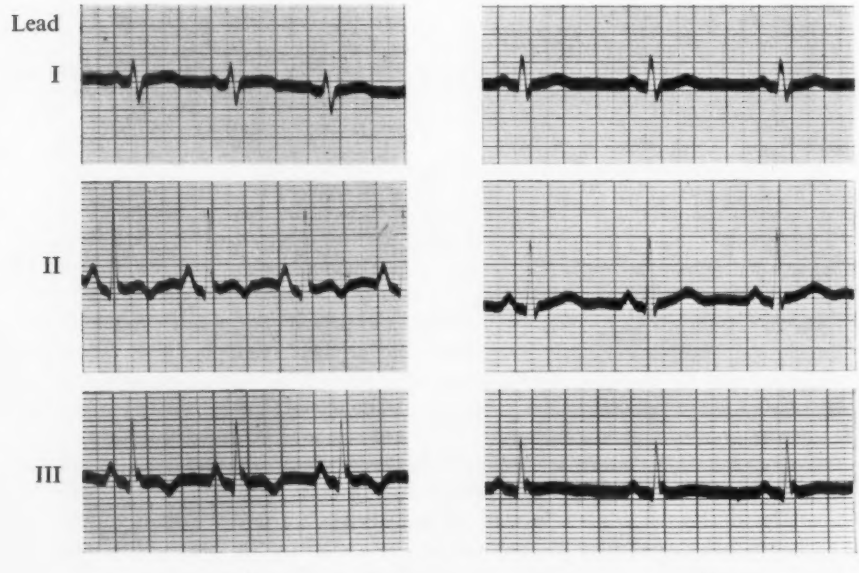

FIG. 3.—Electrocardiographic changes upon change in position from sitting to supine in the case of a young girl of 18, tall and thin, without evidence of heart disease.
(A) Sitting. (B) Recumbent.

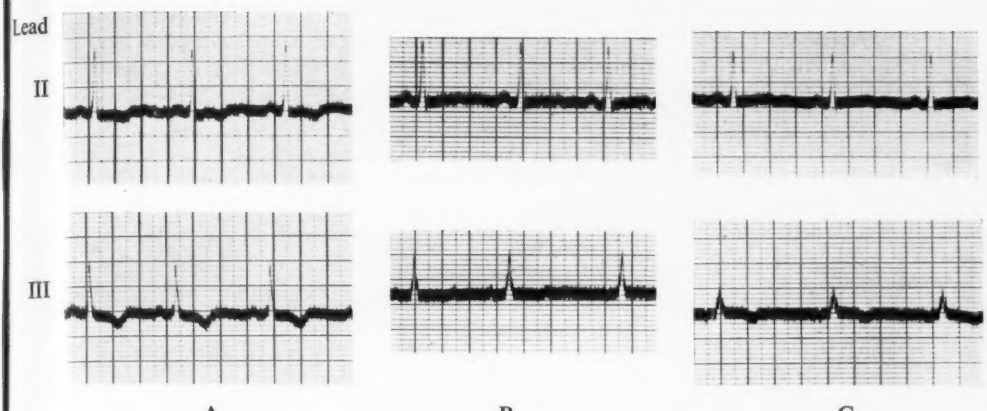

FIG. 4.—Electrocardiographic changes in leads II and III upon change of position from sitting to supine and upon full expiration, in the case of a boy of 15, tall and thin, with a normal heart, vertical in position.

(A) Sitting, during quiet respiration. (B) Recumbent. (C) Sitting, at end of full expiration.

inversion of the T wave is due to a change in posture and therefore a change in contact between the heart and the neighbouring tissues."

DISCUSSION

Lowering, flattening, notching, and inversion of the T waves in lead II are the result of a variety of causes, mostly diseases involving the heart itself. The majority of these are well recognized, ranging from the common changes due to coronary heart disease to the rare findings in myxœdema. Pericarditis, acute and chronic, is an occasional but important cause, acting doubtless through the subjacent myocardial involvement. Myocarditis of rheumatic or other rarer nature can cause such T wave changes, as can also the toxic effects associated with severe infections, renal toxemias and drug action, particularly the well-known intoxication by digitalis. Pronounced enlargement and strain of the right ventricle, acute or chronic, as in the cor pulmonale or in marked mitral stenosis, may cause diphasic flattening or inversion of the T waves in lead II as well as of those in lead III.

The abnormal causes of the changes in T in lead II listed in the last paragraph have been recognized for the most part for years, but interestingly enough the normal or physiological factors responsible for very similar changes have only begun to receive recognition long overdue. Even as recently as 1935 Leimdörfer, as stated above, assumed that such changes without evidence of heart disease or intoxication must of necessity mean that heart disease, particularly coronary insufficiency, is none the less present. Now we are learning better.

There are several physiological causes for changes of T in lead II, but these have not yet been clearly separated by the few writers who have paid any attention to the subject, and in all probability two or more factors are not rarely superimposed. Autonomic nervous influences (preponderantly sympathetic effects) from any cause (exercise, fever, or excitement) with resulting sino-auricular tachycardia, and perhaps also even paroxymal auricular tachycardia, is frequently—in fact usually—attended by a lowering of the T waves in lead II, in contrast to the increase in the height of the T waves that occurs when the vagal action becomes preponderant as during the slowing of the heart following exercise, a well-known effect. Other examples of causes of this autonomic nervous influence are thyrotoxicosis, which we recognized in 1935, and vagal paralysis by atropine, and probably also the toxic effect of tobacco which two of us (P.D.W. and A.G.) described with Starr in 1938. Fright, with its marked nervous reflex effect, is probably another cause for change in the T waves of this type, as will be reported later by ourselves. Thus the presence of considerable tachycardia should make one suspicious of the possible or probable action of this factor when the T waves are low or even inverted in lead II. The sitting and particularly the standing position favours the development of a relative tachycardia compared with the heart rate in recumbency, and so this naturally may be one reason, but we believe not the major reason, for the change of the T waves in lead II with change in posture. Barker and his associates (1939) have also observed significant lowering of the T waves of the electrocardiogram resulting from overventilation with alkalosis, which, as a symptom of anxiety or hysteria, is met with occasionally. (See addendum on p. 240.)

It is, however, the change in position of the heart in the thorax, which occurs particularly in the case of the vertical or drop heart with the subject in the standing or sitting position, that concerns us particularly here. The T waves in lead II are often simply lower than usual, but sometimes they are flat or diphasic, and occasionally they are actually inverted with a simulation of a diseased state, particularly coronary heart disease. The lateness of the inversion of the T waves adds considerably to this simulation of a disease pattern. There is, however, one T wave pattern, namely, that pictured in Fig. 5, that is almost if not wholly pathognomonic of this positional effect, duplicated rarely if at all by pericarditis or coronary heart disease: that is a late slight notching of the T wave, the notch sometimes scarcely depressing the baseline below its zero position. This finding has been an important clue for us in a number of cases.

It behoves us, therefore, when we encounter flattening, notching, or inversion of the T waves in lead II in apparently healthy persons, especially in those who are young and of a slim build with vertical hearts (the very type who are prone to develop neurocirculatory asthenia and anxiety with overventilation) to take electrocardiograms with the subject in the recumbent (supine) posture as well as sitting, and to be sure that the subject is composed and not excited or fearful. It is also always important to record the position of the body when electrocardiograms are taken for the sake of present and future reference, because

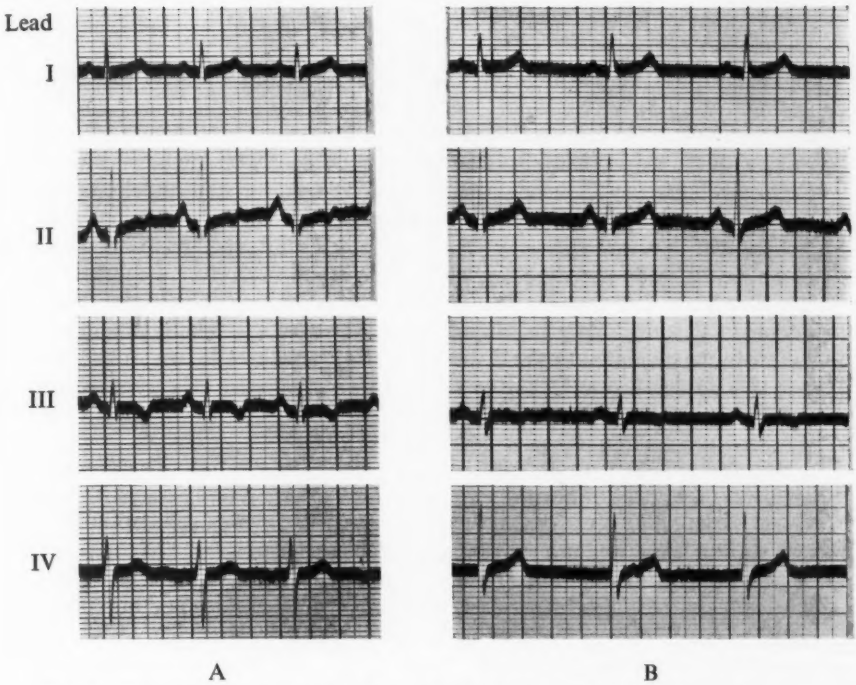

FIG. 5.—Electrocardiographic changes upon change in position from sitting to supine in the case of a woman 45 years old of average build without evidence of heart disease.
(A) Sitting. (B) Recumbent.

of the possible errors in interpretation that may easily arise especially when comparing a series of electrocardiograms. A simple way of recording the position accurately is to draw a straight line which shows whether the thorax is horizontal, vertical, or at a midway angle, thus — | ╲.

Also we should recognize that in these cases posture alone is not the whole answer. It is the position of the heart that counts, and, as has been shown by Scherf and Weissberg and by ourselves, the height of the diaphragm is often very significant, its elevation at full expiration tending to " correct " the T waves, though often not so completely as does recumbency.

Follow-up studies during the past few years, in some cases now extending back nearly ten years, have supported our conviction that the changes we have described in the T waves of lead II are not the result of disease. Our cases have not developed any evidence of abnormality of the heart, and a few who were frail and nervous (with tachycardia) when first seen have grown more robust and composed with improved health in the course of years, and have manifested a " correction " of the T wave variations even in the sitting position; it is probable that a raising of the height of the diaphragm and a decrease in sympathetic overstimulation and hyperventilation account for this

" correction " which was cited but not explained by two of us (P.D.W. and A.G.) in 1935. Also it is, of course, very significant that our most striking cases have been asthenic *young people*.

Finally, this " normal inversion " of the T waves in lead II is quite a common finding. We now recognize several new cases every month in our routine electrocardiographic interpretations and a number of these have been referred to us with the diagnosis of heart disease, often obscure, on the basis of these electrocardiographic findings alone, or with the addition of neurocirculatory asthenia or a physiological heart murmur. Orthostatic hypotension, with tachycardia, in a rare case may be attended by significant T wave inversion, as we have already noted above; whether or not in such cases a decrease in coronary blood flow enters in as a factor we cannot say. It should be realized, of course, that there may be a combination of physiological and pathological factors in the alteration of the T waves in lead II in the same case; a tall lean person may have pericarditis or coronary heart disease or nervous overventilating.

Summary

Inversion of the T waves in lead II of the electrocardiogram, although most commonly the result of heart disease or toxic states, may be a normal physiological variation in occasional persons, particularly those of asthenic habitus with vertical hearts and prone to neurocirculatory asthenia.

The position of the heart is the most important factor in producing this T wave inversion which is found in the sitting or standing position but is corrected by recumbency or by elevating the diaphragm as at full expiration. Autonomic nervous influences comprise another factor, although less striking as a rule, the low or inverted T waves then being attended by tachycardia; any cause of such stimulation, for example, excitement can then be responsible. Fear and anxiety may act through the production of overventilation with resultant alkalosis. Both heart position and nervous influences may be active in the same case.

The relatively common occurrence normally of inversion of the T waves in lead II makes it imperative to recognize its existence in order to avoid erroneous diagnoses of heart disease.

Addendum.—Finally, Mainzer and Krause (1940), described the influence of fear on the electrocardiogram. Immediately before the induction of general anaesthesia, electrocardiographic changes were noted in about two-fifths of 53 cases ; these changes consisted chiefly of S-T depression and lowering or inversion of the T waves similar to that appearing in coronary insufficiency and ascribed by the authors to neurogenic influences reducing the coronary flow.

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PULMONARY VENOUS RETURN VIA THE SUPERIOR VENA CAVA

BY

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From the City General Hospital, Leicester

Received September 16, 1941

The following case is reported as it shows an abnormality which, so far as I am aware, has not been recorded previously. In Abbott's *Atlas of Congenital Cardiac Disease*, 1936, no such case is described, the most closely related one showing pulmonary return into the right auricle via a dilated coronary sinus.

History of the Case

The infant, a female, was born on March 21, 1941. During labour Dr. Hallum, who was in attendance, noted that there was some irregularity of the fœtal heart when bleeding occurred due to a lateral placenta prævia, the os being three quarters dilated. The weight at birth was 6 lb. 11 oz. and the condition was good, although the mother later recalled having been told by the nurse that the child's heart was not right. This observant nurse remains in obscurity. The feet were blue and remained so. When discharged nine days later it is recorded that no murmur was audible and there had been no attacks of cyanosis while in the nursing home.

At home breast feeding was stopped, due to the mother's having " insufficient milk," but bottle feeding was never satisfactory. She vomited practically every time after having taken half the feed, and would become white, with her lips slightly blue or even black. In her ninth week during bathing she appeared breathless and dead beat, but there was no colour change. One day she had a bluish patch on her left cheek. In two months she was only 20 oz. above her birth weight, and was losing. She was taken to a welfare centre, where she was used as a model for a gas-mask demonstration ; immediately after this she was very pale, and remained so until admitted to hospital two days later.

Clinical Findings and Course in Hospital

She entered hospital on May 27, 1941, as a case of inanition for investigation being then nine weeks old.

She was small and undernourished, with marked cyanosis of the extremities,

slight cyanosis of the lips, and a high colour of the rest of the body. Her cry was feeble and almost aphonic. Respirations were 48 per minute, but were not laboured. There was reduplication of the first apical sound and some enlargement of the heart. The liver was palpable and enlarged. When crying, the right side of the mouth was drawn down more than the left. The pulse was regular ; there were no thrills and no pulsation, except slightly at the apex ; there was no bulging of the chest, and the fingers were not clubbed, nor was there tortuosity of the vessels of the eye. The heart rate was regular and 150 per minute ; there was sinus rhythm with no sinus arrythmia. An electrocardiogram showed right axis deviation, large P waves especially in lead II, and inversion of T in leads II and III : these findings suggested auricular and right ventricular preponderance.

The heart was enlarged, and the radiologist reported : " One cannot

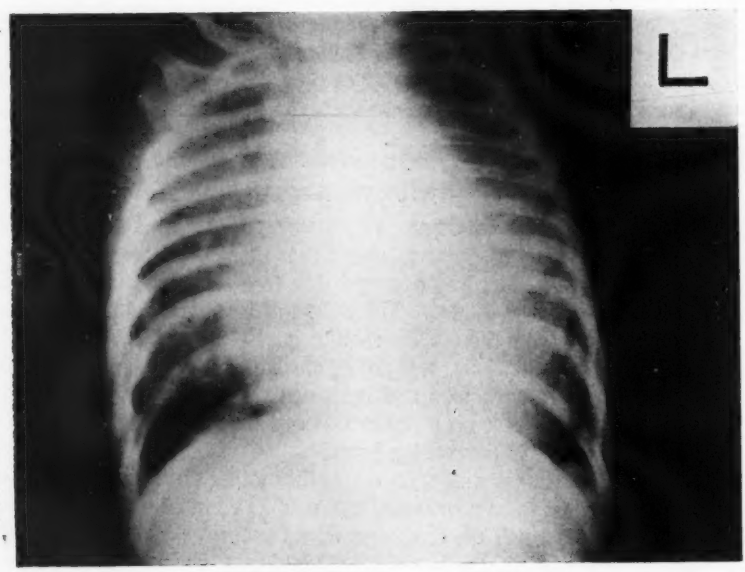

FIG. 1.—Radiogram of the heart taken at 6 feet.

exclude a congenital lesion, but there is no definite evidence of its existence." The hæmoglobin was 118 per cent., and the red blood cells 5,410,000 per c.mm. While blood was being taken for examination she became very cyanosed, almost black, and lay back exhausted ; her breathing was not more distressed than normal. After ten to fifteen minutes she gradually became better.

For the first six days feeding was difficult, as she became exhausted, and, on most occasions, would regurgitate half the feed. Cyanosis was not increased. Regurgitation did not occur after this date, although feeding remained difficult. On one occasion after a feed she became purple and distressed. The apex

rate was rapid and regular. Oxygen through a nasal catheter appeared to cause some improvement.

By the ninth day the cyanosis had largely disappeared, although the respiratory rate remained about 60 per minute. The temperature rose to 99·5° F., while the pulse fell to 130 per minute. There were numerous crepitations in both lungs, with diminution of breath sounds and dullness on percussion over the left lung. Sulphapyridine 0·5 g. was given four-hourly. In place of the reduplicated apical first sound there was now a long, coarse, rumbling systolic murmur of fairly low pitch.

On the tenth day her colour was almost normal. The lungs remained very moist and the pyrexia slight. Immediately after the 6 p.m. feed her face became a greyish blue, her lips and ears were black, her face was twitching and her eyes squinting. For some hours she remained grey and exhausted ; her pulse was regular. On the eleventh day her breathing became very distressed, although there was no cyanosis. The appearances were suggestive of laryngeal or bronchial obstruction. She died at 5.30 a.m.

The circulation is shown diagrammatically in Fig. 2.

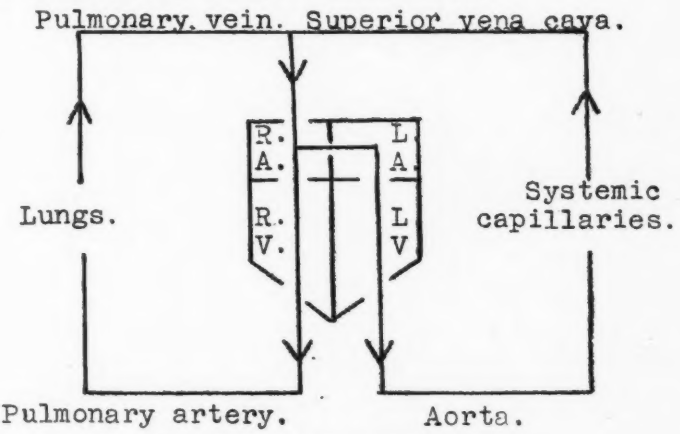

FIG. 2.—Diagram of the abnormal circulation.

POST-MORTEM FINDINGS

There were several ounces of straw-coloured fluid in the pericardial and pleural sacs. The upper two to three feet of the jejunum contained about two ounces of blood mixed with partly digested food. The intestine was very congested. There was no blood in the stomach. The lungs showed mild bronchitis. Macroscopically there were no other abnormalities apart from the heart.

The heart was much enlarged, owing to hypertrophy of the right side, both auricle and ventricle. The heart weighed 52 g. The lungs were joined together by a single pulmonary vein, and from the middle of this arose a thick trunk which united it to the lower and posterior part of the superior vena cava

below the entry of the vena azygos. The pulmonary vein had no direct communication with the left auricle. The hypertrophied right auricle was thus receiving both arterial and venous blood. Many of these points can be well seen in Fig. 3. The sinus venarum was partly separated from the auricle proper by large valves of the superior and inferior vena cava. The pulmonary artery was of great size (about 32 mm. in circumference). The only means of communication between the two sides of the heart was through a small opening (about 11 mm. in circumference) in the upper part of the interauricular septum. This was, presumably, the foramen ovale, despite its high position. The left heart was insignificant in size, forming about one-eighth of the volume of the right heart. The only entry into the auricle was through the opening described. The mitral valve was small but otherwise normal. The aorta was hypoplastic (about 18 mm. in circumference). The ductus arteriosus had closed and was represented by a fibrous cord. Many of these points can be seen in Fig. 4.

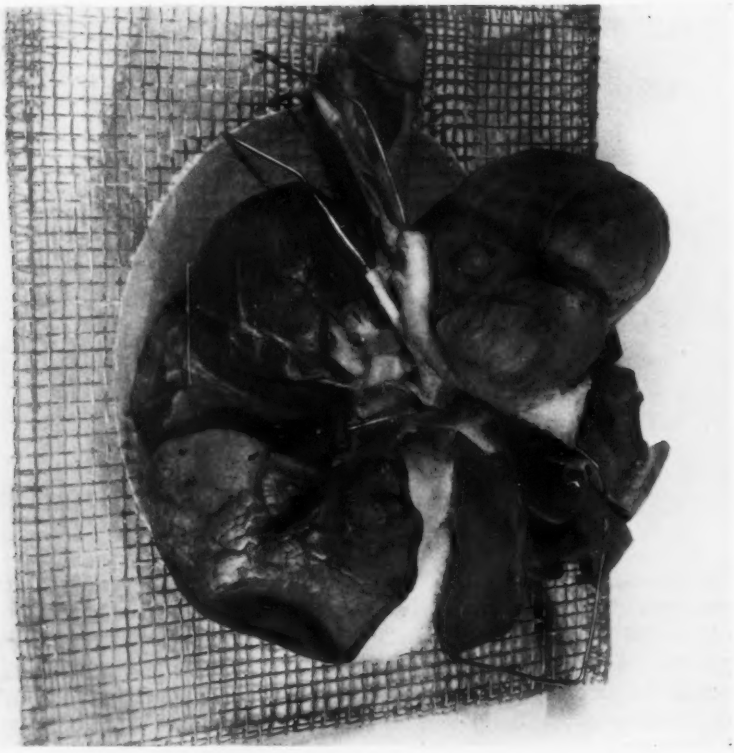

FIG. 3.—The anterior aspect of the preparation. The heart has been turned up and to the left so that only the hypertrophied right auricle and part of the right ventricle are seen. The single pulmonary vein is opened longitudinally, and its connection by a thick trunk with the superior vena cava is shown.

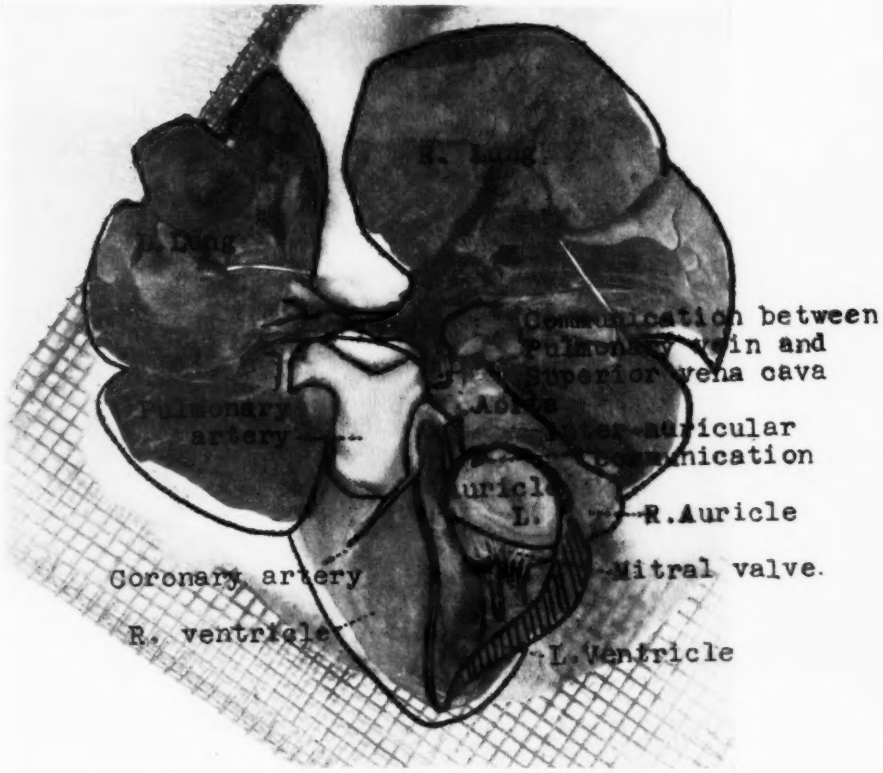

FIG. 4.—The posterior aspect of the heart. The lungs have been turned up. The left auricle and ventricle have been fully opened up to show their minute size. The small aorta and the dilated pulmonary vein are in apposition. Above is the single pulmonary vein and its communication with the superior vena cava.

COMMENTS

It appears that during the course of development the trunk formed from the pulmonary veins which enters the middle of the sinus venosus retained connection with the part of the sinus that ultimately becomes the superior vena cava. The part of the sinus venosus that forms the coronary sinus had separated normally. This arrangement would cause no embarrassment to the fœtal circulation, but with the closure of the placental circulation there was admixture of arterial and venous blood, with a consequent strain on the heart. Persistence of a patent ductus arteriosus would not have helped much, as this would have added to the work of the right ventricle.

Despite the persistent venous shunt, the heart maintained life for eleven weeks. Slight exertion was sufficient to produce embarrassment and cyanosis. It is strange that cyanosis was not manifest all the time, and that increased

pulmonary resistance, due to bronchitis, instead of increasing it caused a diminution.

The heart murmurs are difficult to explain. The apparent reduplication of the first apical sound which was heard on admission may have been due to asynchronous ventricular contraction, but this is not supported by the electrocardiogram. The subsequent change in the murmur, with the development of increased pulmonary pressure, and the magnitude of the P wave suggest that it may have been due to auricular systole.

Summary

A case is described in which the only method by which blood could return from the lungs to the heart was through an opening into the superior vena cava. Communication with the left side of the heart was maintained by a patent foramen ovale.

It is believed that this abnormality has not been recorded previously.

I wish to thank Dr. Page, Medical Superintendent, for permission to publish this case. I also wish to thank Dr. Braithwaite, Consulting Physician, and Dr. Ward, Pathologist to the City General Hospital, Leicester, for their help, and Mr. Calder, senior Radiographer to the Royal Infirmary, Leicester, for taking the photographs.

Editorial Note

A case, somewhat related, has been described by T. B. Johnston (1915) *J. Anat. and Phys.*, **49**, 182. The left pulmonary vein opened into the superior vena cava, but the pulmonary vein on the right side was normal. The left pulmonary vein passed upwards (left superior vena cava) and joined the left innominate vein, which passed across to join the superior vena cava.

CHEST LEAD (CR_1) ELECTROCARDIOGRAMS IN AURICULAR FIBRILLATION

BY

WILLIAM EVANS

From the Cardiac Department of the London Hospita.

Received October 7, 1941

Lewis (1910) was the first to demonstrate the value of chest leads in displaying the movements of the auricle in auricular fibrillation. A trial of five different leads convinced him that those taken over the right auricle showed maximal auricular oscillations and minimal ventricular complexes which facilitated a study of the auricular waves. In a case of complete heart block Cohn and Lewis (1913) found that oscillations from auricular action were greatest when leads were taken directly from the chest wall, the electrodes being placed in the region of the right auricle. Drury and Iliescu (1921) tried two chest leads in 24 patients with auricular fibrillation. In their first or sternal lead an electrode over the second rib on the right side was paired with one over the seventh costal cartilage on the same side, and in their second or antero-posterior lead an electrode at the centre of the sternum was paired with one at the level of the inferior angle of the scapula, two inches to the right of the spine. They favoured the antero-posterior lead. Holzmann (1937) said that abnormalities of the P wave in the electrocardiogram were best recorded by placing the exploring electrode over the right auricle. Lian and Pinchenzon (1938, 1940) used a chest lead with one electrode over the manubrium and the other in the fifth right intercostal space for the investigation of auricular rhythm, and named it the "precordial auricular lead S5." The application of this lead in one case of arrhythmia, which was thought to be auricular fibrillation, gave a tracing exhibiting large auricular waves. They described the condition as auricular tremulation and maintained that it was intermediary between auricular flutter and fibrillation. They postulated that in fibrillation both the duration and shape of the auricular complex in the cardiogram varied, in auricular flutter there was no variation, and in tremulation it was slight. It was while studying the form of the P wave in the chest lead cardiogram CR_1 * in various cardiac disorders that I became interested in tracings obtained from patients with auricular fibrillation. This lead was

* This designation was suggested by the committee reporting on the standardization of precordial leads (*Amer. Heart J.*, 1938, **15**, 235). C is an abbreviation for chest, R for right arm, and the subscript 1 denotes the position of the precordial electrode, namely in the fourth intercostal space at the right border of the sternum.

then recorded in 60 patients in whom clinical examination and the limb lead cardiogram had provided the diagnosis of fibrillation.

It is known that the auricular movements in fibrillation are never faithfully portrayed in the limb lead cardiogram ; the tracing may show no oscillations, or if it does the curves are not constant and vary even in the same patient from insignificant waves to prominent ones, these showing the greatest prominence in one or other lead. When the limb lead cardiograms from the 60 patients were examined for auricular waves they were absent in 28 ; in the remaining 32 the curves were better displayed in leads III and II than in lead I. Actually the oscillations were moderately prominent in lead III in 24 cases, in lead II in 20, and in lead I in 10 cases. Even when distinct coarse waves are seen in limb lead cardiograms they are distorted by movements from sources other than the auricle. In the CR_1 cardiogram extraneous somatic waves are more completely eliminated while the ventricular complexes are small, but its chief advantage over the limb leads appears to be its specificity for recording auricular movements, owing to the plane which the lead traverses. Fig. 1–4 and many

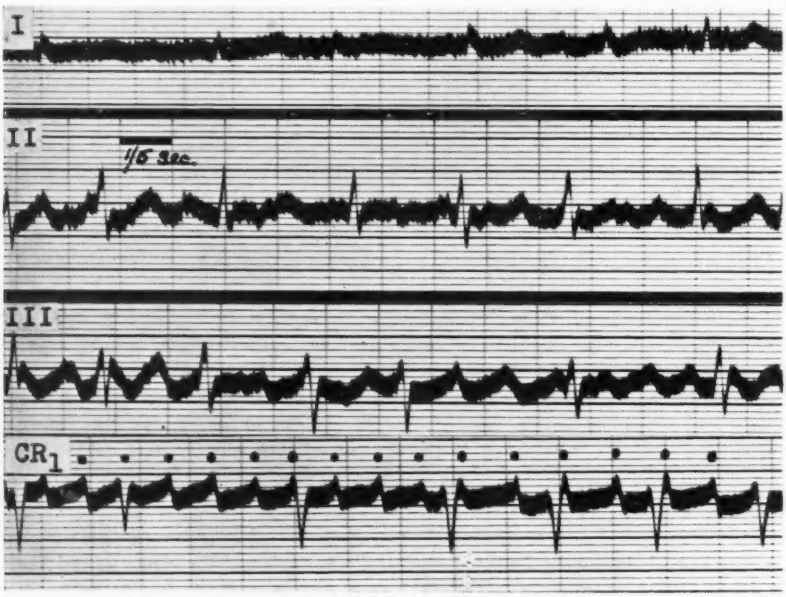

FIG. 1.—Case 29. Auricular fibrillation without apparent cause. Female, aged 64. Auricular rate, 375 a minute. In this and some of the other figures dots denote the summits of the auricular waves.

of the subsequent figures illustrate this. None the less, in many patients this chest lead fails to display the auricular wave to much better advantage than the limb lead. Again, the periodic variation in the amplitude of the oscillations which is characteristic of the limb lead tracings may also be a feature of many of the curves obtained when leading direct from the chest.

In this series 39 CR_1 cardiograms showed large auricular waves (Type I) ; in the remaining 21 the waves were not seen to much better advantage than in the limb lead cardiogram (Type II).

FIG. 2.—Case 22. Auricular fibrillation and mitral stenosis. Female, aged 29. Auricular rate, 384 a minute.

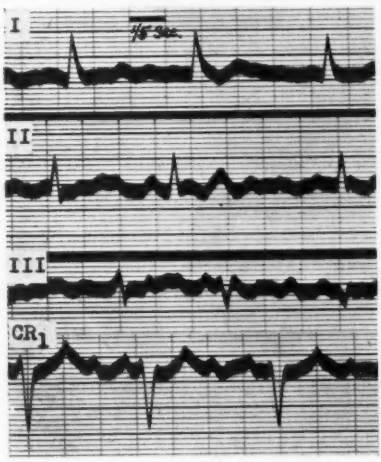

FIG. 3.—Case 25. Auricular fibrillation without apparent cause. Male, aged 53. Auricular rate, 375 a minute.

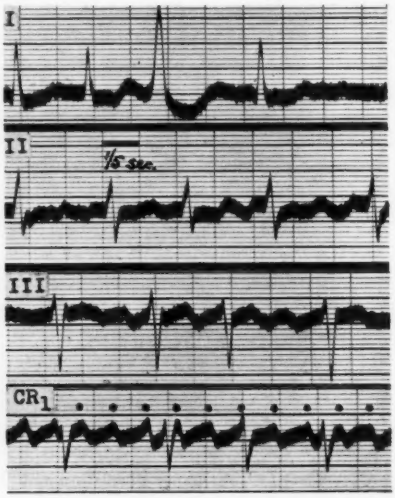

FIG. 4.—Case 32. Auricular fibrillation and mitral stenosis. Female, aged 50. Auricular rate, 362 a minute.

Large Auricular Waves in CR_1 : Type I

Although 39 of the 60 patients have been included in this group, 2 (Cases 38 and 39) are kept apart from the remaining 37 and will be discussed at the end of the section.

A description of the auricular wave in the CR_1 cardiogram should relate to its *form*, *rhythm*, and *frequency*, and these features of the curve will now be considered separately.

Form of the Auricular Wave

The form of the auricular wave in the CR_1 cardiogram varied from patient to patient, but it remained fairly constant in the same individual with each curve differing slightly from the other. It did not show that regularity of pattern characteristic of the auricular flutter curve, but there was a close resemblance. In most tracings the fibrillation waves presented a gradual upstroke followed by a rather steep downstroke. The curves were written in continuity and without a pause at the iso-electric level, proving that the auricle was at no time electrically quiescent. Further typical illustrations are given in Fig. 5–9. Often the amplitude of the waves became subject to a phasic variation, from time to time increasing and diminishing in height. During a period when the amplitude of the oscillation diminished (see end of CR_1 tracing in Fig. 5) the wave often became bifid, giving the appearance of two

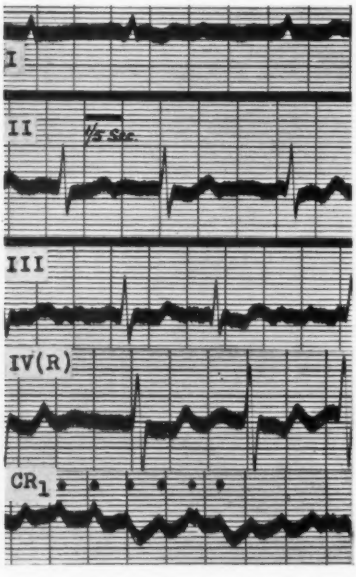

Fig. 5.—Case 17. Auricular fibrillation and mitral stenosis. Male, aged 44. Auricular rate, 394 a minute. Bifid auricular wave at end of CR_1 lead.

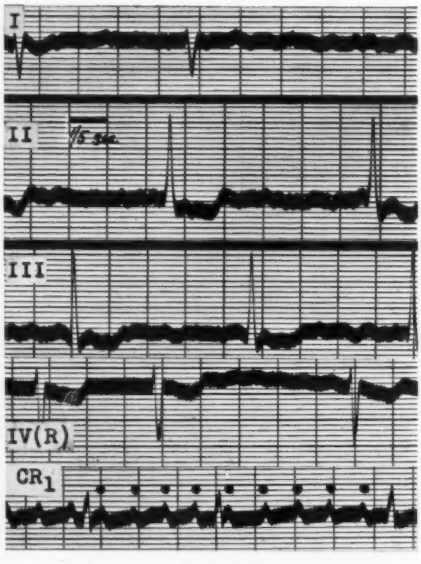

Fig. 6.—Case 31. Auricular fibrillation and mitral stenosis. Female, aged 22. Auricular rate, 363 a minute.

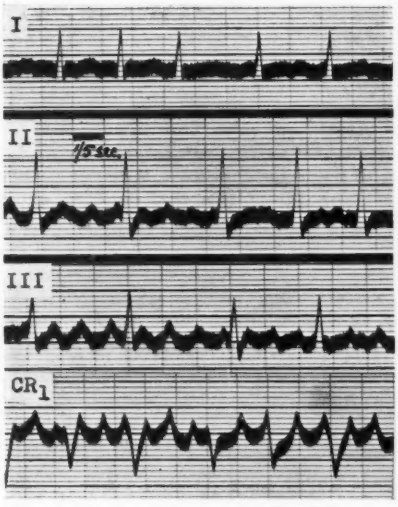

FIG. 7.—Case 36. Auricular fibrillation and thyroid toxæmia. Male, aged 55. Auricular rate, 350 a minute.

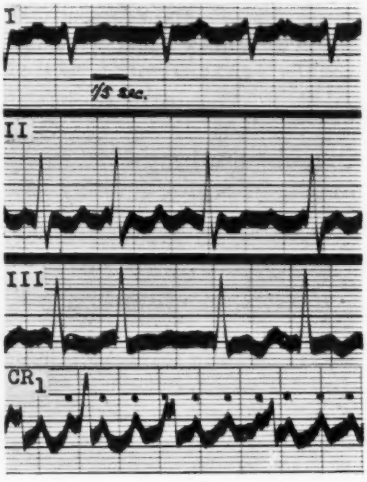

FIG. 8.—Case 18. Auricular fibrillation and mitral stenosis. Female, aged 25. Auricular rate, 394 a minute.

separate waves. No extrinsic cause for this variation could be made out ; the respiratory movements, for instance, did not influence the amplitude, and the same observation was made by Drury and Iliescu (1921). When comparing five different chest leads, Lewis (1910) stated that the oscillations were conspicuous or the reverse according as the contacts were close to or distant from the right auricle, but that whenever conspicuous oscillations were obtained, then inconspicuous oscillations appearing in part of the tracing were an expression of some intrinsic variation in the auricle. In my cases the amplitude of the fibrillation wave did not depend on the condition initiating the arrhythmia nor on the size of the right auricle, for the waves were no larger in patients with mitral stenosis than in those without, who were seen on radioscopy to have a heart unaffected in shape or in size (Fig. 1 and 3).

Rhythm of the Auricular Wave

Describing the oscillations that replace the normal P waves when auricular fibrillation sets in, Lewis (1925) stated that they were never quite regular in form, amplitude, or length. The 37 patients in this series may be divided into three groups according to the rhythm of the fibrillation waves in the CR_1 electrocardiogram. There were 18 patients in the largest group where the rhythm was irregular ; even in these the irregularity was not obvious at first and only emerged when measurements were taken. It was difficult to determine the influence of the ventricular systole or diastole on the auricular rhythm in these cases. In the second group of 13 patients the rhythm of the auricular

waves was regular except when it was disturbed (occasionally, frequently, or constantly) by ventricular systole; in 7 the auricular wave succeeding ventricular systole was delayed, and in 6 it was hastened. This finding is in disagreement with that of Drury and Iliescu (1921), who stated that both ventricular systole and diastole were without effect on the auricular oscillations. The third group held 6 patients in whom the rhythm of the auricular waves was regular and was undisturbed by ventricular systole ; in these patients although the auricular rhythm was regular and very like auricular flutter, the shape of the waves differed slightly from beat to beat.

Frequency of the Auricular Wave

Different rates have been assigned to auricular contraction in auricular fibrillation by different writers. Thus, Battro (1937) mentions 400 to 600

TABLE I

37 PATIENTS WITH LARGE AURICULAR WAVES IN THE CR_1 CARDIOGRAM (TYPE I)

Case No.	Age	Cause of Fibrillation	Digitalis or No Digitalis	Average Ventricular Rate	Auricular Rate	Limb leads showing Fibrillation Waves
1	64	No cause found	No D.	100	488	II and III
2	63	Mitral stenosis	No D.	100	453	0
3	60	No cause found	D.	100	450	II and III
4	43	Mitral stenosis	D.	90	450	0
5	34	No cause found	No D.	85	450	III and II
6	30	Mitral stenosis	D.	37	437	0
7	45	do.	D.	75	436	II and III
8	45	do.	D.	85	428	0
9	39	do.	D.	130	428	II and III
10	33	do.	D.	70	420	I
11	26	do.	D.	30	416	0
12	68	Hypertension	No D.	150	409	II
13	38	Mitral stenosis	No D.	92	400	III and II
14	58	No cause found	No D.	130	400	III
15	63	Hypertension	D.	90	400	0
16	46	Mitral stenosis	D.	60	400	III and II
17	44	do.	No D.	100	394	0
18	25	do.	D.	120	394	II
19	29	do.	D.	55	390	I
20	37	do.	D.	100	388	I
21	76	Hypertension	No D.	75	385	III
22	29	Mitral stenosis	D.	100	384	0
23	66	do.	D.	130	375	II and III
24	53	do.	D.	66	375	0
25	53	No cause found	No D.	85	375	III and II
26	38	Mitral stenosis	D.	85	375	II
27	31	do.	D.	130	375	0
28	39	do.	D.	120	375	I, II, and III
29	64	No cause found	No D.	120	375	III and II
30	51	Mitral stenosis	D.	64	375	II and III
31	22	do.	D.	85	363	0
32	50	do.	D.	150	362	II and III
33	43	do.	D.	75	362	II
34	39	do.	D.	72	355	0
35	27	do.	D.	66	353	I and III
36	55	Thyroid toxæmia	No D.	130	350	III and II
37	49	Mitral stenosis	D.	75	350	I

and Bramwell (1932) 500 to 600. Drury and Iliescu (1921) found the auricular rate to be 390 to 588 in twenty-four cases (chest leads), with an average of 473. Herrmann (1936) stated that it was 450 to 600, Levine (1936) 400 or more, Lewis (1925) rarely below 400 and rarely above 600, Pardee (1928) 350 to 500, Wenckebach and Winterberg (1927) 434 in one patient with chest lead, and White (1937) gave the figure as 400 to 600. The disparity in the figures is explained by the fact that the observations have been based in most instances on the frequency of the auricular waves in the limb lead cardiogram. Such observations are necessarily precarious, owing to the imperfections of this electrocardiogram in showing the complete details of auricular contraction. Thus, in calculating the rate of the main movement of the auricle both the coarse and the fine oscillations have been separately regarded as depicting auricular contraction. This impression of greater frequency created by notching or splitting of the main auricular wave is illustrated towards the end of the CR_1 tracing in Fig. 5. Again, even if the oscillations in the limb lead cardiogram are moderately distinctive for a moment calculation of the auricular rate based on a few beats will be inaccurate. Table I shows the remarkable constancy of the rate of auricular contraction in patients with auricular fibrillation.

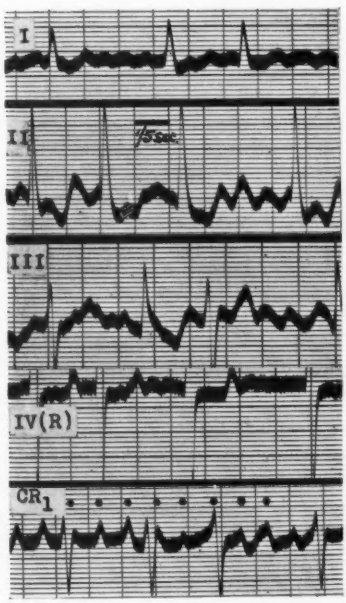

FIG. 9.—Case 9. Auricular fibrillation and mitral stenosis. Female, aged 39. Auricular rate 428 a minute.

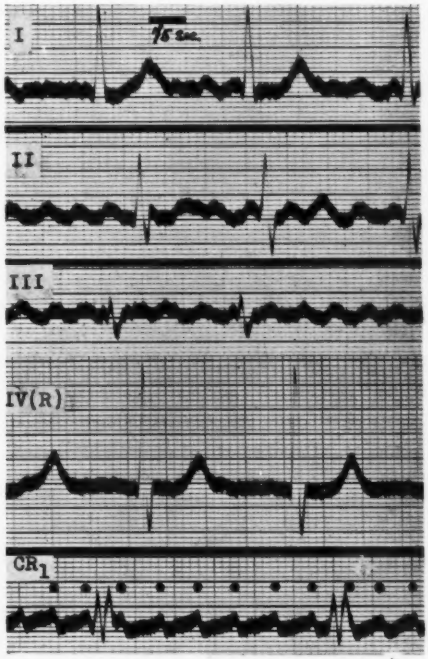

FIG. 10.—Case 38. Mitral stenosis. Male, aged 43. Auricular rate, 312 a minute. Limb leads suggest auricular fibrillation. The CR_1 lead shows auricular flutter (see text p. 254).

More than half had an auricular rate of 375 to 400 a minute ; with two exceptions the rate was 350 to 450, and the average rate was 400 for the 37 patients. The auricular rate did not depend on the ventricular rate, for a patient showing the highest auricular rate (488) had an average ventricular rate of 100, while a patient with the lowest auricular rate (350) had a ventricular rate of 130. Digitalis was found to have no effect on the auricular rate in patients re-examined with the CR_1 electrocardiogram during a period of digitalis therapy. Quinidine did slow the auricular rate (see Fig. 12).

Two patients (Cases 38 and 39) were regarded clinically as auricular fibrillation and mitral stenosis. In Case 38 the limb lead cardiograms supported this view, although the tracing in some ways resembled flutter : the CR_1 cardiogram, however, gave the diagnosis of auricular flutter (Fig. 10). In Case 39 the ventricular complexes were infrequent and usually irregular, and no auricular waves were visible ; in all, 15 limb lead cardiograms were recorded, and they appeared to support the diagnosis of auricular fibrillation. The CR_1 cardiogram still left doubt about the exact diagnosis of the type of auricular activity but showed 2 : 1 auriculo-ventricular block (Fig. 11). This last tracing shows a rhythm similar to that found in chest lead cardiograms in paroxysmal tachycardia, which are being investigated.

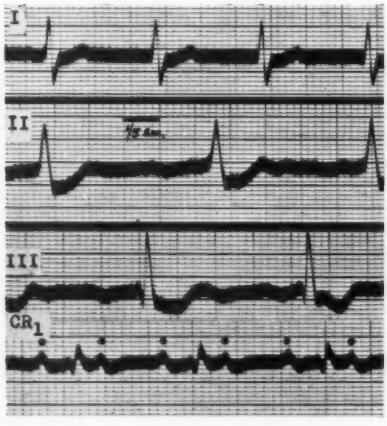

FIG. 11.—Case 39. Mitral stenosis. Female, aged 52. Limb leads suggest auricular fibrillation, but CR_1 shows that whatever the auricular rhythm may be there is 2 : 1 auriculo-ventricular block. Auricular rate, 210 a minute.

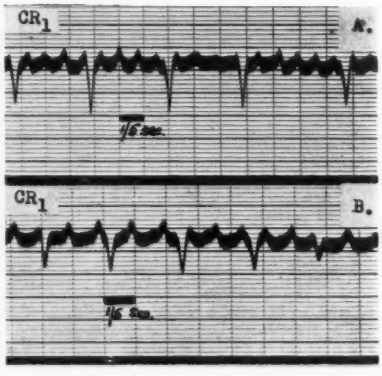

FIG. 12.—Slowing of the auricular rate by quinidine. The tracing above was taken before, and that below after, a week on quinidine (9 grains daily). The auricular rate was reduced from 375 to 340 a minute.

SMALL AURICULAR WAVES IN CR_1 : TYPE II

In most of the 21 patients of this group the CR_1 cardiogram demonstrated auricular waves which were more conspicuous than those recorded in the limb lead cardiogram, but the curves were neither prominent nor constant enough to permit a study of the form of individual waves nor to allow an

estimate of the rate of auricular contraction. An attempt was made to discover some factor that might determine whether a patient with auricular fibrillation would show large (Type I) or small (Type II) auricular movements in the CR_1 cardiogram. To this end special attention was paid to the cause and duration of the arrhythmia, the ventricular rate, digitalis influence, the size of the right auricle, presence of " fibrillation " waves in the limb lead cardiogram, thickness of the chest wall, and to the possibility of a faulty technique in applying the chest electrode.

The *cause* of the arrhythmia appeared to have no influence in deciding the amplitude of the auricular wave in the CR_1 cardiogram. In the favourable series (Type I) of 37 patients, mitral stenosis was the cause in 27, hypertension in 3, thyroid toxæmia in 1, and cause unknown in 6. In the unfavourable series (Type II) of 21 patients, mitral stenosis was the cause in 18, and the etiology was unknown in 3.

An attempt was made to ascertain the *duration* of the fibrillation in each patient. From this inquiry the prominence of the auricular wave did not appear to depend on the duration of fibrillation ; the arrhythmia appeared to be recent (within three months) in 14 of 37 Type I cases and in 6 of 21 Type II cases.

TABLE II

21 PATIENTS WITH SMALL AURICULAR WAVES IN THE CR_1 CARDIOGRAM (TYPE II)

Case No.	Age	Cause of Fibrillation	Digitalis or No Digitalis	Average Ventricular Rate	Limb leads showing Fibrillation Waves
1	60	No cause found	No D.	60	0
2	48	Mitral stenosis	No D.	85	III and I
3	39	do.	D.	60	0
4	34	do.	D.	80	0
5	55	do.	D.	94	0
6	21	do.	No D.	42	0
7	54	do.	D.	60	0
8	44	do.	D.	55	0
9	51	do.	D.	50	0
10	45	do.	D.	85	0
11	39	do.	D.	42	0
12	58	do.	D.	90	0
13	48	do.	D.	50	0
14	42	do.	D.	55	0
15	48	do.	D.	90	III and I
16	68	No cause found	D.	85	III
17	60	do.	D.	85	0
18	34	Mitral stenosis	D.	42	I and III
19	58	No cause found	No D.	66	III
20	58	Mitral stenosis	D.	46	0
21	44	do.	D.	60	III, II, and I

When the *ventricular rate* was compared in patients in the two groups a difference was observed. It was much more rapid in those with prominent auricular waves, averaging 92 per minute for the 37 patients (see Table I), compared with 66 for the 21 patients with inconspicuous waves (see Table 2).

Again, whereas no Type II patient had a rate higher than 94, in Type I patients it was over 100 per minute in 17. Nevertheless, although conspicuous auricular waves were always present when the ventricular rate was rapid, they were sometimes observed with a moderately slow ventricular rate (Fig. 3).

The inquiry on rate led to an investigation of the incidence of *digitalization* in patients in each group. Although it was a little commoner in Type II patients, the influence of digitalis on the auricular wave was not obvious.

It was natural to assume that in Type I patients a projection of the *right auricle* beyond the right border of the sternum favoured the recording of auricular movements by an electrode placed in this area. Lewis (1910), when advocating the more frequent use of chest leads in the study of oscillations produced by the fibrillating auricle, stated that the best curves were obtained in cases in which the right auricle was distended, as in mitral stenosis, when a large part of its surface lay in contact with the chest wall. The present investigation does not support this view, for it showed that conspicuous auricular waves in the CR_1 electrocardiogram did not depend on extension of the heart to the right. Thus, most of the patients were examined by radioscopy, and in 15 patients showing conspicuous waves enlargement of the right auricle was either absent or slight, whereas in 9 patients showing inconspicuous waves right auricular enlargement was either moderate or considerable.

That patients with prominent fibrillation waves in the limb lead electrocardiogram would also show conspicuous waves in the CR_1 was expected, and this proved to be the case, so that coarse auricular oscillations were seen in one or more limb leads in 25 of the 37 Type I cases and they were absent in 15 of the 21 Type II cases. At the same time the exceptions emphasize that this criterion is not a constant guide in predicting the two types.

The thickness of the chest wall did not affect the amplitude of the auricular waves in the CR_1 electrocardiogram, and that inconspicuous curves were not the result of a faulty technique in applying the chest electrode was proved by recalling patients of both series and obtaining in them tracings identical with the previous records.

Summary and Conclusions

The chest electrocardiogram, CR_1, was taken in 60 patients with auricular fibrillation in order to study the auricular wave. Large and conspicuous waves, resembling those of auricular flutter, were found in 39 patients (Type I). In the remaining 21 cases (Type II) the auricular waves were small and no more conspicuous than in many limb lead electrocardiograms.

No factor was discovered that determined in a patient with auricular fibrillation whether the auricular movements would be represented in the CR_1 cardiogram by large or by small waves, except that if the ventricular rate was high and the auricular oscillations were conspicuous in the limb lead cardiogram, distinct waves were likely to show in the CR_1 cardiogram. The ætiology and the duration of the arrhythmia, the size of the right auricle, digitalization

or its absence, and the thickness of the chest wall, did not seem to decide whether a patient would exhibit large (Type I) or small (Type II) auricular waves.

The features of the auricular waves were clearly shown in those patients classified as Type I, and these were specially studied. The *form* of the wave, varying slightly in different patients, resembled the waves found in auricular flutter, although lacking the same constant pattern. The waves recurred continuously without a pause at the iso-electric level, and each showed a gradual upstroke followed by a steeper downstroke. The amplitude of the wave was occasionally subject to a natural variation, and this was independent of any known extrinsic factor, including respiration. Small waves were often bifid, thereby giving the appearance of two waves and explaining the more frequent oscillations seen in the limb lead cardiogram. The wave was no larger in mitral stenosis than in non-rheumatic auricular fibrillation.

The *rhythm* of the auricular waves was slightly irregular in 18 patients, regular in 13 except for interruptions by ventricular systole when the succeeding auricular wave was as often delayed as quickened, and quite regular in 6 cases, where the rhythm was undisturbed even by ventricular systole, so that the tracing closely resembled that of auricular flutter.

The *frequency* of auricular waves in auricular fibrillation was found to be reasonably constant ; the rate was 375 to 400 per minute in more than half the cases ; with two exceptions the range was 350 to 450 and the average rate for all patients was 400. Higher rates of auricular contraction generally assigned to auricular fibrillation have been based on observations on limb lead cardiograms which often contain more than one oscillation representing a single beat of the chest lead cardiogram. The rate was uninfluenced by digitalis but it was reduced by quinidine.

In two patients in whom a clinical diagnosis of auricular fibrillation was supported by a limb lead cardiogram, the CR_1 tracing demonstrated auricular flutter in one and 2 : 1 auriculo-ventricular block in the other. The value of this chest lead is also seen in elucidating cases of so-called impure flutter, better regarded as fibrillation, and in the interpretation of records with auricular waves in the limb lead cardiogram sometimes subdued or obscured, as in heart block and in paroxysmal tachycardia.

The chest, CR_1, cardiogram permits the limited definition of *auricular fibrillation* as a succession of waves at a rate of about 400 per minute, differing slightly in size and shape, and with a rhythm almost as often regular as irregular. The ventricular responses in fibrillation have no constant relationship to the auricular waves, probably because the auricular rate is too high for any regular control.

In contrast *auricular flutter* shows a succession of waves at a rate of about 300 per minute, identical in size and shape, and perfect in rhythm. The ventricular responses in flutter are determined by the auricular waves giving a fixed or varying ratio.

I wish to thank Dr. John Parkinson, Physician to the Cardiac Department of the London Hospital, for his helpful criticism of this paper.

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PROCEEDINGS OF THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

The FIFTH ANNUAL GENERAL MEETING of the Cardiac Society of Great Britain and Ireland was held at the School of Geography, Oxford, on Friday, April 18, 1941.

CHAIRMAN: A. G. GIBSON

36 Members and 8 Visitors were present
The Chairman took the chair at 10.15 a.m.

PRIVATE BUSINESS

1. The minutes of the last meeting were read and confirmed.
2. The accounts, audited by Evan Bedford and Curtis Bain, were presented by the Council and approved, the balance being £66 4*s.* 11*d.*
3. The Secretary proposed on behalf of the Council and the Society approved the following addition to Rule 11
   " In exceptional circumstances the Council may release a Member from this rule."
4. The Chairman proposed on behalf of the Council, and the Society agreed
   " that in view of the importance of keeping the Society in being during the war, Rule 23 should be in abeyance and that the Secretary should be asked to continue in office, although he would have completed five years after the 1941 meeting."
5. On the recommendation of the Council, it was decided to ask the following to accept nomination as Honorary Members: J. B. Herrick, Chicago, U.S.A., and Frank N. Wilson, Ann Arbor, U.S.A.
6. Five Associate Members were elected Ordinary Members; two new Associate Members were elected; and ten Associate Members were re-elected for another period of three years.
7. Crighton Bramwell, Manchester, and Henry Moore, Dublin, were elected members of the Council for the years 1941–45.
8. The Secretary reported that:—
   (1) since the last meeting the Council had heard with regret of the death of three Honorary Members—
   Maud Abbot of Canada,
   Karel Frederik Wenckebach of Vienna, and
   Charles Laubry of Paris;
   (2) the Committee appointed at the request of the Ministry of Labour and National Service to advise on the instructions for the examination of the cardiovascular system of recruits had reported to the Ministry, and that the report had been accepted in part;
   (3) the Council had appointed a Committee to consider the question of members from other parts of the British Empire;
   (4) the Journal Board of the British Medical Association had written expressing

their satisfaction with the continuation of the British Heart Journal under the difficult conditions of the War; and

(5) the Council had appointed Shirley Smith as Assistant Secretary for another year and wished to thank him for his services during the last year.

Discussion on Early Signs of Cardiovascular Disease (*Morning Session*)

Chamberlain opened the discussion on the early signs of *valvular* heart disease. Auscultation was still the method of diagnosis of paramount importance, diastolic murmurs in aortic regurgitation and mitral stenosis establishing the diagnosis with certainty in most cases. There were certain other clinical, electrocardiographic, and radiological signs, some of which in combination might be sufficient to establish a diagnosis of valvular disease without the characteristic murmur.

In *mitral stenosis* the clinical signs usually emphasized were accentuation or reduplication of the mitral first, pulmonary second, or mitral second sounds. An abrupt loud mitral first was the most constant associated clinical sign in mitral stenosis and was of more importance when the heart rate was slow. Accentuation of the pulmonary second was a much less constant phenomenon in early cases of mitral stenosis though common in the more advanced ones, whilst accentuation of the mitral second was rather uncommon. Reduplication, though common in the pulmonary second, was not a constant or a particularly helpful sign. The electrocardiogram frequently showed no changes, but a split P wave, especially of low voltage, was such a common sign in mitral stenosis that it was worthy of consideration in doubtful cases, especially when associated with the less common right ventricular preponderance.

Enlargement of the left auricle, seen best on screening in the right (I) oblique view after a barium swallow, and prominence of the pulmonary arc were important radiological signs; the latter was an earlier and more constant sign than auricular enlargement.

The diagnosis of *aortic stenosis* was debateable. Some held that the characteristic anacrotic pulse associated with a harsh systolic murmur (with or without a thrill) over the aortic area and conducted into the neck was essential for diagnosis. For others a harsh murmur and thrill were alone acceptable. Some of the latter cases proved eventually to be due to genuine stenosis and when left ventricular hypertrophy was present the presumptive diagnosis might be justified even in the absence of an anacrotic pulse, for such a pulse indicated a well-developed aortic stenosis.

For the diagnosis of *aortic regurgitation* auscultation still remained the principal method. The basal diastolic murmur might long precede other clinical or instrumental methods of diagnosis. The aortic second sound was rarely abolished until regurgitation was free and the vascular phenomena were late in appearance.

Terence East discussed the early signs of *hyperpietic* disease.

Changes in the arterial system and in the heart could only be regarded as late signs, so the early diagnosis would depend on the interpretation of the readings of the sphygmomanometer.

The chief difficulty lay in distinguishing the transient rise in blood pressure due to nervousness from the true early high blood pressure. On the whole the diastolic pressure was a safer guide than the systolic because it was less labile. Nervousness might cause rise in pulse rate and not in pressure, and vice versa. The diastolic pressure might sometimes be unreadable. In doubtful cases raising the arm might give a clearer diastolic reading. In any borderline case the more readings the better. Even quite a long time spent waiting recumbent would not always bring a nervous rise down. The way the heart beats was perhaps the most constant indication of the

degree of nervousness. In borderline cases suspicions of hypertension might be confirmed by overweight, by the personal appearance, and by the family history. If 160 to 150 systolic and 95 to 100 diastolic were taken as the border zone, the interpretation of the figures would depend on as many other factors as possible, and must often remain a matter of opinion, the correctness of which only time would show.

BRUCE PERRY spoke on the early signs of *myocardial* disease.

The early signs of damage to the myocardium varied considerably according to the ætiology of the myocardial lesion. In diphtheria, conduction defects were often the first evidence of pathological processes. In acute rheumatism, the mitral regurgitant murmur due to the dilated mitral ring was the usual first evidence of myocarditis followed rapidly by evidence of cardiac enlargement. In the other types of heart disease, thyrotoxic, hypertensive, and senile, the first evidences of involvement of the myocardium were usually the symptoms of cardiac insufficiency. Here the importance of dyspnœa was paramount, and it became of considerable value to be able to distinguish dyspnœa arising from cardiac insufficiency from that resulting from other causes. An estimation of the arm–tongue circulation time had proved of considerable value in this, though this was generally an indication of the onset of failure. The great difficulty in accepting minor changes in the electrocardiogram or in the X-ray picture of the heart as early evidence of myocardial damage lay in the wide variation met with under normal conditions.

SHORT COMMUNICATIONS

THE RIGHT PECTORAL ELECTROCARDIOGRAM IN AURICULAR FIBRILLATION

WILLIAM EVANS showed how well the auricular movements in auricular fibrillation are displayed in the right pectoral electrocardiogram. When this lead was recorded in 42 cases of auricular fibrillation conspicuous auricular waves were seen in 25, but in the remaining 17 patients they were not seen to better advantage in this lead than in limb leads.

A study of the favourable tracings showed that the form of the auricular waves, although not so completely regular as the waves of flutter, was remarkably uniform. In more than one-half the cases the wave recurred regularly, and in the others the irregularity was slight; it occurred at intervals and was sometimes associated with ventricular systole. The rate of auricular contraction in the majority was slower than 400 a minute which was the average for all the cases. Occasionally the right pectoral lead showed typical flutter in patients previously regarded as instances of fibrillation following clinical examination and limb-lead electrocardiography. Further the application of this chest lead in patients believed to have simple paroxysmal tachycardia will sometimes demonstrate the arrhythmia to be auricular flutter, and an example of this was exhibited.

(Published in full on p. 247)

THE COLLATERAL CIRCULATION IN COARCTATION OF THE AORTA

CRIGHTON BRAMWELL and A. MORGAN JONES

(Published in full: see p. 205)

THE HÆMIC FACTOR IN ANGINA PECTORIS

B. T. PARSONS-SMITH described some cases illustrating the hæmic factor in angina pectoris, and discussed the factors involved.

Coronary sclerosis was the basis of myocardial ischæmia in 80–90 per cent of

cases. Myocardial anoxæmia might be produced also by various types of anæmia and possibly also by polycythæmia, and these syndromes might be curable by treatment. He recounted the details of three patients suffering from chronic hæmorrhagic anæmia, hypochromic anæmia, and pernicious anæmia. In the last case anginal symptoms had developed, and in each case the repair of the anæmia had abolished the angina. The importance of blood counts in difficult cases of angina pectoris was illustrated by these clinical examples. The existence of pernicious anæmia might remain unsuspected until the later stages of its development.

The Electrocardiogram of the Stokes-Adams Attack

John Parkinson and Cornelio Papp (*introduced*)

(Published in full: see p. 171)

"Chronic Myocarditis"

John Hay urged that the words "chronic myocarditis" should not be used as a diagnosis because he considered it misleading. Myocarditis should signify an inflammation of the myocardium, and admittedly there were sound reasons for the term chronic rheumatic myocarditis when referring to the advanced phases of a rheumatic infection.

In the later decades of life, however, myocardial fibrosis was circulatory in origin and degenerative rather than inflammatory. He feared that the teachers in medicine and the writers of text-books were very largely responsible for the persistence of this term and quoted several examples from well-known books, illustrating the loose and confusing use of the term. The pathologists were also to blame; one referred to "so-called fibrous myocarditis." Another wrote that obliteration of a branch or branches of a coronary artery was the "most common cause of cardiac fibrosis, or as it is usual to term it 'interstitial myocarditis.'" Similarly in books dealing with electrocardiography, one finds "Myocarditis, Chronic" used almost as an equivalent of "Myocardial Disease" or "Myocardial Damage."

There was no need to multiply instances. It seemed quite clear that one writer after another had timidly refused to discard this unsatisfactory nomenclature because others were equally timid.

It might be true that in the production of ischæmic fibrosis changes occurred that might legitimately be termed inflammatory, but he was convinced that to call such a pathological product myocarditis was to mislead. Cardio-sclerosis was a clearer and more appropriate term.

He felt that the Society as a whole should refrain from perpetuating such a muddling and misleading phrase as chronic myocarditis, except in those cases such as rheumatic carditis, where it might be justified.

Discussion on the Treatment of Coronary Thrombosis (*Afternoon Session*)

The speakers had been asked to consider if the orthodox treatment was all essential and based on sound principles.

Curtis Bain introduced the discussion and suggested that the period of rest in bed might be reduced to three weeks, provided that

(*a*) the patient had had no previous attacks;

(*b*) there had been no significant fall in blood pressure, no fever, and no pericarditis; and

(*c*) there were no complications. He recognised that this suggestion would be provocative of discussion.

For treatment of the painful phase intravenous morphia was valuable; it would

stop the pain in about 3 minutes. One sixth of a grain should be injected intravenously, followed at once by one quarter hypodermically and later by another quarter, if required. Other drugs included theophylline-ethylene-diamine (Cardophyllin). It could be given intravenously in emergencies such as pulmonary œdema or complete heart block. Although useless in true shock following coronary thrombosis, respiratory stimulants such as coramine might revive certain patients who passed into a condition of pseudo-shock. *Oxygen therapy* was of undoubted use in those cases where the pain had persisted in spite of adequate doses of morphia and in those in whom cyanosis developed at any stage of the illness. The small open-top type of oxygen tent were well tolerated.

Digitalis should be reserved for those cases in whom cardiac failure with œdema developed. Auricular fibrillation was paroxysmal in type and the paroxysms usually subsided naturally without causing difficulty. In severe cases fibrillation might be present from the outset, but in these it was doubtful whether a myocardial poison such as quinidine would not do more harm than good.

The *dietetic regime of under-nourishment* was introduced by Masters. It aimed at reducing the work of the heart by lowering the basal metabolic rate with a concomitant fall in the pulse rate. Upward pressure from flatulent distension of the stomach was avoided. The diet was maintained for the whole period during which the patient was in bed and increased gradually thereafter. The diet suggested by Masters had been found to be rather severe, but a diet consisting of carbohydrates 100 g., proteins 45 g., and fats 35 g. has been well tolerated, and the results from it had been satisfactory.

JOHN HAY visualized three stages in the treatment of coronary thrombosis. The first was the stage of onset with pain and shock. The second or intermediate stage was that of healing. Thirdly, at the end of three or four months the softened necrosed area had been replaced by scar tissue, and this was the period in which the cardiac reserve had to be built up and the patients' limitations defined.

A few days after the onset the pain was in abeyance, and the symptoms of shock had disappeared and the patient—when at rest—was comfortable, though often apprehensive.

As the production of a sound scar takes at least three months, so the patients' activities must be supervised for that period and kept well within safe limits, more especially in the first few weeks.

To obtain a leisurely heart rate with no sudden stresses or cardiovascular excitement (leaving to nature the business of repair), mental excitement, restlessness and apprehension are to be mitigated; veganin, theominal, or bromides are useful in producing mental calm, and a more philosophic outlook. For the first three or four days the patient needs only fluids such as weak tea, orange juice, lemonade, and drinks containing glucose or honey. Later, a light mixed diet can be given, but each meal should be small and appetising, and any food tending to produce flatulence avoided.

For the first two weeks the patient should be fed by the nurse. If pyrexia is present or if *congestive cardiac failure* has developed the nourishment should be limited to one and a half pints of skimmed milk per diem, and as the indications of cardiac failure diminished a gradual return to light, mixed, easily digested meals was indicated.

The fairly large doses of morphia required to relieve the pain at the onset, and the excessive sweating would almost certainly cause *constipation* and upset the digestion, but it was generally agreed that it is best not to worry the patient with laxatives or enemata until the third or fourth day. At the same time it was important to avoid flatulent distension; a tight belly hampering the diaphragm was an unfair and avoidable handicap to a disabled heart.

In the later stages of the illness from three months onward when the patient would be gradually finding his feet, a new difficulty might present itself, namely, the danger

of his becoming unduly apprehensive about his cardiac condition and drifting into a condition of invalidism. Conversely, the active type of patient, resenting restrictions, might return prematurely to full work and run the risk of overstraining his damaged myocardium with disastrous results.

He agreed with Bain as to the value of oxygen in treatment. Drugs had their place in the alleviation of pain, as mental sedatives, and in the treatment of cardiac failure if and when it appears; but the essential fact to be borne in mind is, that beyond everything, physical rest is the most important factor in enabling the damaged heart to recover.

GILCHRIST (*in absentia*) said that for the relief of pain and for the promotion of mental and physical rest there was no drug to compare with full, and if necessary repeated, doses of morphia. In spite of the relief of pain some patients surviving the initial infarct died in the course of a few days from shock. This aspect of treatment has tended to be neglected, and though there was no specific remedy for this peripheral vascular failure it is possible that more might be done to safeguard the patient. On the other hand, a very large number of patients did very well, after the pain has been relieved, with no active drug treatment. Complete rest in bed might do much more than it was sometimes given credit for, particularly if the rest in bed were complete in every detail.

In the severer cases of shock there was, for instance, a natural hesitation to employ intravenous saline solutions and transfusions of whole blood, although these were commonly given in shock from other causes. In the early stages a plentiful supply of hot water-bottles or preferably a " shock cage ", fitted up with more than the usual electric bulbs and continued for 24 or 48 hours, should be provided. Clinical studies in other related forms of peripheral vascular failure indicated that benefit was likely to be obtained from the use of the active principle of the suprarenal cortex. It had been used after extensive burning injuries and in virtue of its power to regulate water-balance and cellular permeability, a potent extract of suprarenal cortex was worthy of a more extended trial in the severer cases of cardiac infarction. The value of intravenous therapy was doubtful, but concentrated blood serum might be preferable to massive saline infusions or whole blood transfusions.

The development of an abnormal rhythm such as a ventricular tachycardia, with the subsequent development of ventricular fibrillation, was probably more common than supposed, particularly as the patient himself might be unaware of any change in heart rhythm. Quinidine might be used as a preventative measure when the indications were those of an extensive infarct: 0·2 g. at eight-hour intervals was harmless and might be of real benefit. The continuous drip method for the intravenous administration of quinidine as proposed and used by Hepburn and Rykert, was valuable in the treatment of paroxysmal tachycardia. By vigorous shaking, 50 to 60 grains of quinidine sulphate could be dissolved in 500 c.c. of 5 per cent glucose solution; after filtering and warming, the sterile solution should be run at a rate of 1 to 2 c.c. per minute until normal rhythm returns or until cinchonism is induced.

* * * * *

After luncheon Dr. H. W. Garrod showed the Society round the College, and in particular the Library where there were interesting books and records of Harvey.

Dinner was served in Merton Hall, through the kindness of the Warden, Sir John Miles. Fraser proposed the health of the Society and recalled the early history of the Cardiac Club, founded after the last war.

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